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American Veterinary Medical Association

FORMERLY AMERICAN VETERINARY REVIEW

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KANSAS CITY ESTABLISHES RECORD

The unexpected happened at Kansas City. All attendance figures for previous A. V. M. A. conventions were surpassed. Contrary to expectations, veterinarians poured into the convention city, accompanied by their wives and children, until the turnstiles at the registration desk stopped clicking at the 1450 mark, some forty more than were registered at Minneapolis in 1929, and over a hundred more than at Detroit in 1930. Had it not been an off year, it is quite possible that Kansas City would have put the attendance mark at 2,000 or higher.

Although the convention did not officially get under way until Tuesday morning, over 300 were registered by Monday evening. The various committee meetings in the morning, the Executive Board meeting in the afternoon, and the conference of delegates from state and provincial associations in the evening accounted for the large number of advance registrations. Incidentally, these lightened the load on the efficient staff from the Kansas City Convention and Visitors Bureau, at the registration desk on Tuesday morning, when the meeting opened.

The 1931 convention will go down in A. V. M. A. history as one of the "smoothest running" meetings in the 68 years of the organization's existence. Sessions were started on schedule and

were adjourned the same way. President Hall kept things moving at the general sessions and the section officers did the same at the nine sectional meetings held Wednesday and Thursday mornings. In this way the official program was adhered to from start to finish, with only a few minor changes.

The address of President Hall was the subject of much favorable comment. It would be out of the question to discuss it adequately here, and it is suggested that every member read the address when published in the JOURNAL. Treasurer Jacob was able to report the Association in a good financial position, in spite of a slightly decreased income the past year. The report of the Secretary showed that the membership had been maintained at approximately the 1930 figure, in the face of a falling off in the number of new members, as well as a heavy loss the result of deaths.

Atlanta, Georgia, was selected for the 1932 meeting, although Havana, Cuba, made a strong bid for the privilege of entertaining the Association next year. We have met in southern territory on only a few occasions, and the choice of Atlanta apparently was popular with practically everybody in attendance at Kansas City. Chicago, Illinois, and Winnipeg, Manitoba, put in bids for 1933. The Association approved a recommendation of the Executive Board that the 1934 International Veterinary Congress be held in Philadelphia, which, in effect, means that the A. V. M. A. convention that year will be held in the City of Brotherly Love.

A contest for the office of president of the A. V. M. A. made it necessary to try out the new plan of balloting for officers, and as a result the new A. V. M. A. ballot-box was used for the first time at Kansas City. The election machinery ran smoothly and only favorable comments were heard. Everybody at the meeting on Wednesday had a chance to vote and 409 took advantage of the opportunity. The count of the ballots showed Dr. R. R. Dykstra, of Manhattan, Kansas, a winner over Dr. N. F. Williams, of Fort Worth, Texas. This is the first time that the Sunflower State has been honored with the presidency, but the tenth time during the past quarter-century that the Association has selected its leader from the field of veterinary education.

The five vice-presidents were elected by acclamation. Dr. Clodoaldo Antonio Arias y Delgado, of Havana, Cuba, was named first vice-president, thereby complimenting our sister republic, the Pearl of the Antilles. Dr. J. T. Glennon, of Newark,

N. J., was reëlected second vice-president, in recognition of the field of general practice. An Army officer, in the person of Major Harry J. Juzek, was elected third vice-president. Education and research was recognized in the selection of Dr. I. E. Newsom, of Fort Collins, Colo., as fourth vice-president, and the federal Bureau of Animal Industry was complimented by the naming of Dr. J. S. Healy, of Madison, Wis., as fifth vice-president. Dr. M. Jacob, of Knoxville, Tennessee, was reëlected treasurer to serve his fourteenth successive term.

The alumni meetings Tuesday evening were well attended. The various alumni groups returned to the old plan of meeting separately, and the Hotel Baltimore was taxed to the limit to provide accommodations for the different colleges represented. We hope to publish reports of these meetings next month. The banquet Wednesday evening was attended by over 500, and was the occasion for a number of fine addresses and some spicy entertainment. Dr. H. D. Bergman, of Ames, Iowa, in the capacity of toastmaster, and Dr. J. L. Jones, of Blackburn, Mo., as chairman of the Banquet Committee, kept things moving at a lively pace all through the evening.

The all-day clinic on Friday was a fitting climax to a very eventful week. Dr. S. L. Stewart, of Olathe, Kansas, had prepared one of the most elebarate clinic programs ever presented. It was divided into six sections which were kept going simultaneously after the preliminary general clinic was held. This was featured by a demonstration of the DeLaval Magnetic Combine milking system and an address by Dr. O. C. Murphy, Milk Commissioner, Kansas City Board of Health. Dr. J. S. Jenisen, B. A. I. inspector-in-charge at Kansas City, put on a pathological exhibit that was unusual in showing the great variety of diseases and abnormal conditions detected in inspecting meat and meat food products.

A buffet lunch was served the 800 veterinarians at the clinic by Wilson and Company. Dr. F. A. Imler was master of ceremonies on this occasion and his staff consisted of about a dozen officials of Wilson and Company, in the persons of Messrs. Bickett, Chappell, Corrigan, Dever, Donmeyer, Gherrity, Hagedorn, Keller, Reavis, Silverstien and Sopp.

Dr. Ashe Lockhart had his hands full providing accommodations for the unexpectedly large attendance. Incidentally he should be credited with having made good on his promise of comfortable weather, not only in the Pompeian Room at the

Hotel Baltimore but in Kansas City generally. Several times during the week the temperature started to climb but did not get very far. The exhibits were well housed and attracted many visitors. We hope to have a detailed report on the exhibits later.

The local newspapers were quite liberal in giving space to articles covering the convention. It has always been difficult to get what we consider adequate space for abstracts of scientific papers presented at our conventions. In the eyes of the average city editor, these have limited news value, unless they contain something sensational, which is rarely the case. Dr. C. D. Folse was in charge of publicity and is to be complimented for the way in which he handled what is always a difficult assignment.

The entertainment of the ladies, directed by Dr. J. C. Flynn, is being covered in another report. The ladies have become such an important part of our conventions that a separate report seems to be about the only way to do justice to their activities. Many veterinarians at the meeting frankly confessed that their wives were responsible, more than anything else, for their presence in Kansas City. Considering that there were about 400 of the fair sex registered, it will be appreciated what an important role they play.

As chairman of the Committee on Local Arrangements, Dr. A. T. Kinsley was the recipient of unstinted and well-earned praise for both the elaborate preparations for the meeting and the manner in which everything was executed. Like the efficient general, Dr. Kinsley had anticipated almost any contingency that might arise. His men were on the job until the convention came to a close.

In a report of this kind many interesting features necessarily must be passed over rather hurriedly or not even mentioned. In the October issue of the JOURNAL will appear the official proceedings as well as additional comments on various phases of the 68th annual convention of the A. V. M. A.

THE LADIES HAD A FINE TIME

In Minneapolis we ladies had variety for our entertainment, in Los Angeles we had scenery, but in Kansas City we had a most delightful example of perfection in entertaining with purely social functions. Where could we find anything more beautifully planned and executed than the Auxiliary breakfast, unless it was the luncheon at the Athletic Club, or the one in the Pompeian

room at the Baltimore? Not only were we served with delicious foods, but we really were entertained and pleasantly, too, by the variety of programs furnished at each of the functions.

The card party on Wednesday was unique in more ways than one. Someone must have spent many, many hours selecting the beautiful prizes and planning the many minute details necessary for such an event, but most interesting of all was the ladies' quartet, the members of which are daughters of Doctors E. A. and J. R. Shikles, of Dearborn, Mo. Many ladies, though not knowing this connection with the profession, expressed particular pleasure in the program furnished by the ladies' quartet.

The reception and dance this year was specially attractive. Usually some of us rather dread the first half-hour of this annual event, but we had no need to do so this year. It was a grand success. There was none of the usual dragging and slow movement, it went with a good swing from the very first to the very last.

Another thing which seemed quite attractive to us elders this year was the large number of young people in attendance. Certainly it is a good thing for our boys and girls to know one another. It is only in such gatherings as this just past that they can meet their own kind, under such ideal conditions and from such diverse parts of the country.

It would not be right to describe in any way this year's ladies' program and not mention, very definitely, Dr. Flynn and Mrs. Lockhart. We who have had any experience along such lines gasp in wonder when we realize the amount of time and thought and energy necessary to put on such a program. Here's a good rousing cheer for them and for Kansas City 1931. They will long be remembered by those of us who were there.

A. M. H.

APPLICATIONS FOR MEMBERSHIP

(See July, 1931, JOURNAL)

FIRST LISTING

ARMSTRONG, ROBERT P. — Shippensburg, Pa.
B. S., Pennsylvania State College, 1926
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

BEAUMONT, EDWARD V. — 1833 N. 13th, Kansas City, Kans.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: B. F. Gooch and C. B. Clement.

BOLTON, JAMES C. — Lathrop, Mo.
D. V. M., Chicago Veterinary College, 1918
Vouchers: O. E. Herl and H. E. Curry

BOWLER, GEORGE E. Tecumseh, Mich.
D. V. M., Michigan State College, 1930
Vouchers: Lee Davisson and Harold E. Wright.

BURKE, JOHN W. 337 Kenwood Ave., Dayton, Ohio.
D. V. M., Ohio State University, 1923
Vouchers: O. V. Brumley and Leonard W. Goss

CADY, P. L. Arlington, Nebr.
D. V. S., Kansas City Veterinary College, 1910
Vouchers: Chas. D. Folse and C. H. Hays.

CHESHIRE, JOHN B. Hopkins, Mo.
D. V. M., Kansas State College, 1931
Vouchers: Edwin J. Frick and Howard J. Hearrington.

DILLARD, W. C. Farmington, Mo.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: F. H. Suits and E. L. Dicke.

DRAGOO, JAMES A. Connersville, Ind.
M. D. C., Chicago Veterinary College, 1908
Vouchers: R. A. Craig and H. Meade Hamilton.

EISENHOWER, ELMER C. Navarre, Kans.
D. V. S., Kansas City Veterinary College, 1910
Vouchers: J. C. Flynn and C. B. Clement

ELLIOTT, G. H. Bowling Green, Mo.
D. V. S., Kansas City Veterinary College, 1909
Vouchers: Ray O. Porter and Chas. D. Folse.

EMBICK, FRANK J. Laurel, Nebr.
D. V. M., Chicago Veterinary College, 1912
Vouchers: G. G. Miller and Carl J. Norden.

FITCH, HUGH C. Missouri Valley, Iowa.
D. V. M., Iowa State College, 1918
Vouchers: James Morris and H. B. Treman.

GLUHM, CHARLES C. 625 Prytania Ave., Hamilton, Ohio.
D. V. M., Ohio State University, 1931
Vouchers: W. F. Guard and W. R. Krill.

GRIFFIN, JAMES E. 330 Federal Bldg., Madison, Wis.
D. V. M., McKillip Veterinary College, 1918
Vouchers: W. A. Cornell and R. S. Heer.

HADLEY, T. W. 346 State Ave., Kansas City, Kans.
D. V. S., Western Veterinary College, 1900
Vouchers: B. W. Conrad and A. T. Kinsley.

HAUSCHEN, CHESTER A. Brewster, Minn.
D. V. M., Iowa State College, 1926
Vouchers: H. C. H. Kernkamp and N. L. Nelson.

HETH, CHESTER M. 708 N. 3rd St., La Crosse, Wis.
D. V. M., Grand Rapids Veterinary College, 1916
Vouchers: T. H. Ferguson and W. Wisnicky.

HOPPING, J. LEE 1035 Marietta St., Atlanta, Ga.
D. V. M., Ohio State University, 1917
Vouchers: L. A. Mosher and O. V. Brumley.

HURST, HUGH 1351 Murphy Lane, Salt Lake City, Utah
D. V. M., Colorado Agricultural College, 1916
Vouchers: N. C. Spalding and C. T. Jones.

JENNINGS, WM. E. N. Y. State Agr. Exp. Station, Geneva, N. Y.
D. V. M., Cornell University, 1931
Vouchers: W. A. Hagan and E. L. Brunett.

JOHNSON, PAUL A. 2420 Marshall St., Little Rock, Ark.
D. V. M. Ohio State University, 1908
Vouchers: Walter Martin and R. W. Williams.

KRUEGER, H. C. 220 S. Vermont Ave., Mason City, Iowa.
D. V. M., Chicago Veterinary College, 1917
Vouchers: H. E. Curry and H. D. Bergman.

LARSEN, NELSON L. Petersburg, Nebr.
D. V. M., St. Joseph Veterinary College, 1919
Vouchers: Bernhard Witt and Carl J. Norden.

LEENERTS, GEORGE H. Humphrey, Nebr.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: Chas. D. Folse and Darrell E. Trump.

LOGAN, EDWARD A. 2607 Ashland Blvd., Saint Joseph, Mo.
D. V. M., Kansas State College, 1909
Vouchers: J. S. Clark and R. R. Dykstra.

MCINTOSH, H. A. Villisca, Iowa.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: F. D. Weimer and Jesse L. Shabram.

MCTYRE, DOUGLAS W. South Hill, Va.
D. V. M., United States College of Veterinary Surgeons, 1916
Vouchers: Hansford H. Rowe and Taylor P. Rowe.

MOCK, CLARENCE E. Ulysses, Nebr.
D. V. M., Kansas City Veterinary College, 1915
Vouchers: E. C. Jones and A. A. Anderson.

MOHR, CHARLES A. 1616 N. Elwood, Tulsa, Okla.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: C. R. Walter and E. E. Harnden.

MOLT, FRED S. Hinton, Okla.
D. V. M., Indiana Veterinary College, 1912
Vouchers: H. Wood Ayers and C. R. Walter.

NELSON, CONRAD L. 3541 W. 10th St., Oklahoma City, Okla.
D. V. S., Kansas City Veterinary College, 1906
Vouchers: Walter H. Martin and C. R. Walter.

NEVITT, JOHN VICTOR 1706 Mo. Pacific Bldg., Saint Louis, Mo.
B. S. in A. H., Colorado Agricultural College, 1916
M. S., D. V. M., Cornell University, 1924
Vouchers: O. S. Crisler and C. W. McLaughlin.

PARKER, H. C. Hillsdale, N. Y.
D. V. M., Cornell University, 1927
Vouchers: J. G. Wills and Wright J. Smith.

PUGH, JOHN Phillipsburg, Kans.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: Edwin J. Frick and M. L. Dietrich.

RAEBONE, ALEXANDER L. 410 Elmwood Ave., Ithaca, N. Y.
D. V. M., Cornell University, 1931
Vouchers: E. Sunderville and Herbert L. Gilman.

REECE, CHESTER L. 103 S. First, Savannah, Mo.
D. V. S., Saint Joseph Veterinary College, 1911
Vouchers: Ralph Graham and J. R. Shikles.

RICHARDS, HARRY S. 7645 Delmar Blvd., Saint Louis, Mo.
D. V. M., Saint Joseph Veterinary College, 1921
Vouchers: S. W. Haigler and J. T. Jenneman.

RICHELIEU, FRED L. 420 W. Grove St., Visalia, Calif.
B. S., D. V. M., State College of Washington, 1929
Vouchers: R. B. Griffenhagen and C. R. Rey.

ROSE, THOS. P. York, Nebr.
D. V. S., Kansas City Veterinary College, 1910
Vouchers: J. C. Flynn and D. W. Hurst.

RUSSELL, HARRY B. Live Stock Exchange Bldg., South Saint Joseph, Mo.
D. V. M., Kansas City Veterinary College, 1913
Vouchers: J. S. Clark and A. T. Kinsley.

SALL, CARL J. Bertrand, Nebr.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: Carl J. Norden and Geo. A. Young.

SLATER, J. HARVEY Carrollton, Mo.
D. V. S., Kansas City Veterinary College, 1903
Vouchers: A. T. Kinsley and J. D. Ray.

SNYDER, JOHN R. 116 W. Fourth, Gordon, Nebr.
D. V. M., Saint Joseph Veterinary College, 1918
Vouchers: Bernhard Witt and Carl J. Norden.

STEEL, CHARLES E. 1309 Linwood, Oklahoma City, Okla.
D. V. S., Kansas City Veterinary College, 1899
Vouchers: C. H. Fauks and C. R. Walter.

STEWART, LEWIS C. 42nd & State Sts., East Saint Louis, Ill.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: Robt. Rives and S. W. Haigler.

STOCKLER, B. J. Live Stock Exchange Bldg. Annex, South Saint Joseph, Mo.
D. V. M., Ohio State University, 1908
Vouchers: J. S. Clark and O. V. Brumley.

STONE, WILFRED J. R. 1, Joplin, Mo.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: J. D. Ray and E. L. Young.

STUBBS, CHARLES D. Old State House, Little Rock, Ark.
D. V. M., Saint Joseph Veterinary College, 1912
Vouchers: Walter Martin and R. W. Williams.

THOMAS, RAYMOND F. 86 Cain St. N. W., Atlanta, Ga.
D. V. M., Indiana Veterinary College, 1912
Vouchers: J. C. Wright and L. A. Mosher.

WAGAMAN, GROVER M. Kokomo, Ind.
D. V. M., Indiana Veterinary College, 1912
Vouchers: C. C. Donelson and T. A. Sigler.

WEISS, A. H. 821 Florida St., Sabetha, Kans.
D. V. M., St. Joseph Veterinary College, 1920
Vouchers: B. W. Conrad and E. J. Frick.

WERMUTH, JOHN J. Warwick, N. Y.
D. V. M., Cornell University, 1912
Vouchers: J. F. DeVine and George E. Jacobi.

WILSON, DON R. Plattsburg, Mo.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: J. C. Flynn and H. A. Wilson.

WIRTZ, JOHN H. 1604 Euclid Ave., Oklahoma City, Okla.
D. V. M., Chicago Veterinary College, 1920
Vouchers: C. C. Hisel and C. R. Walter.

WILKE, THEODORE E. West Plains, Mo.
D. V. S., Kansas City Veterinary College, 1911
Vouchers: F. H. Suits and J. L. Jones.

WOELFFER, ELMER A. 324 S. Hale St., Wheaton, Ill.
D. V. M., Cornell University, 1931
Vouchers: Harry Caldwell and L. A. Merillat.

ZIEGENBEIN, FRANK E. Nebraska City, Nebr.
D. V. M., Chicago Veterinary College, 1920
Vouchers: W. T. Spencer and R. R. Dykstra.

Applications Pending

SECOND LISTING

Chapman, W. B., Leon, Iowa.
Ellis, Carlton C., 503 Dryden Rd., Ithaca, N. Y.
Huddleson, I. Forest, Michigan State College, East Lansing, Mich.
Kershaw, Cass J., 8043 Wornall Rd., Kansas City, Mo.

McGavick, James A., 514 E. 30th St., Tacoma, Wash.
MacKellar, Robert S., Jr., 329 W. 12th St., New York, N. Y.
Oglesby, W. T., Iowa State College, Ames, Iowa.
Scott, David C., Tekamah, Nebr.
Snyder, G. A., Perry, Iowa.
Ward, R. L., Batavia, Iowa.

COMING VETERINARY MEETINGS

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. September 8, 1931. Dr. C. L. Miller, Secretary, 508 S. Humphrey Ave., Oak Park, Ill.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. September 8, 1931. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. September 16, 1931. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. October 7, 1931. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

Utah Veterinary Medical Association. State Capitol Bldg., Salt Lake City, Utah. October 7, 1931. Dr. E. A. Bundy, Secretary, 1656 25th St., Ogden, Utah.

Eastern Iowa Veterinary Association. Hotel Montrose, Cedar Rapids, Iowa. October 13-14, 1931. Dr. Paul V. Neuzil, Secretary, Blairstown, Iowa.

Maine Veterinary Medical Association. Bangor, Maine. October 14, 1931. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Maine.

American Humane Association. Hotel Pantlind, Grand Rapids, Mich. October 19-22, 1931. J. N. Walker, General Manager, 80 Howard St., Albany, N. Y.

Pennsylvania State Veterinary Medical Association. Laboratory of the Pennsylvania Bureau of Animal Industry, Harrisburg, Pa. October 22-23, 1931. Dr. Harry R. Church, Secretary, Box 403, Harrisburg, Pa.

Florida State Veterinary Medical Association. Orlando, Fla. October 26-27, 1931. Dr. J. V. Knapp, Secretary, Box 445, Tallahassee, Fla.

Keystone Veterinary Medical Association. Philadelphia, Pa. October 28, 1931. Dr. C. S. Parkwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

CORRELATION OF BLOOD REACTIONS WITH THE BREEDING RECORDS OVER A PERIOD OF YEARS IN AN ABORTION-INFECTED HERD OF CATTLE*

By C. F. CLARK, *Department of Animal Pathology*

Michigan Agricultural Experiment Station
East Lansing, Mich.

The data presented in this paper were obtained from a pure-bred dairy herd consisting of from sixty to one hundred animals of breeding age. Bang's disease (infectious abortion) has existed in the herd for the past twenty years. Prior to the time this project was started, the herd had been tested for Bang's disease at irregular intervals but no systematic effort had been made to limit the spread of infection. In March, 1926, the Department of Animal Pathology began the studies herein reported. Monthly agglutination tests have been made of all animals of breeding age. All available fetal membranes have been examined, breeding records recorded and the usual clinical treatment applied to all cases of retained fetal membranes, and other ailments of the genitalia. From March, 1926, to June, 1927, the project was carried on by Dr. E. R. Carlson, at that time a graduate assistant in the Department. From September, 1927, to June, 1929, Dr. D. B. Meyer, a graduate assistant, was in charge of the project. Since July 1, 1929, the author has had charge of the project. The data recorded by Drs. Carlson and Meyer have been used, for which the author herewith makes grateful acknowledgment and assumes complete responsibility for any errors in presentation. The author is indebted to Mr. J. P. Torrey, of the Department of Bacteriology, for supplying the data relative to udder infection, and to Dr. E. T. Hallman for many helpful suggestions.

In compiling data it was decided to select those animals with a minimum of two years of monthly agglutination tests. The one hundred animals with the greatest number of monthly agglutination tests were chosen. No effort has been made to select or to omit any particular animals. These one hundred animals represent 3,700 monthly agglutination tests. The maximum number for any one animal is 48; the minimum is 21. Only five animals have less than 24 reactions recorded.

*Presented at the sixty-seventh annual meeting of the American Veterinary Medical Association, Los Angeles, Calif., August 26-29, 1930. Published with the permission of the Director of the Michigan Agricultural Experiment Station as Journal Article No. 43 (n. s.).

Agglutination Tests

Agglutination tests were made by the rapid method of Huddleson and Abell.[1] From March, 1926, until March, 1928, positive results with the rapid test were checked with the tube test. After the accuracy of the rapid test had been established, the check test was discontinued. Tests were made in dilutions of 1:25, 1:50, 1:100, 1:200 and 1:400. The symbols used to describe reactions are: + = positive agglutination; P = partial agglutination; T = trace agglutination; − = negative. Sera negative in all dilutions or trace in 1:25 are called negative. Positive sera are those positive in 1:100 to higher. Suspicious sera are those with reactions between the above described, *i. e.*, PT, +PT, ++PT.

Animals have been grouped according to their final agglutination reaction. To change to a negative group from a positive or suspicious group, a negative reaction for at least six months was required.

Breeding Records

In the following tables all calves may be considered delivered alive and healthy unless otherwise recorded. In tabulation the

Table I—*Key to grouping of animals*

Animal	Group	Animal	Group	Animal	Group	Animal	Group
19	IIC2	152	IIA2	196	IA	232	IIA3
41	IA	153	IIA1	198	IB	233	IIB1
42	IB	155	IIA2	199	IIA2	235	IIB2
44	IB	159	IIB3	202	IIA1	236	IIA3
47	IIC1	162	IB	203	IIB1	237	IIA3
49	IC	164	IA	204	IIB1	238	IIA3
62	IIC1	166	IIB1	205	IIC2	240	IIC1
64	IIB1	167	IIB3	208	IIB1	241	IIA2
71	IIA1	168	IIB1	209	IIB3	242	IIC1
73	IIA2	171	IID1	210	IIA1	243	IA
83	IIA1	173	IA	211	IB	245	IA
89	IIA3	174	IIA3	214	IIB2	250	IIA1
95	IB	178	IID1	215	IIB1	251	IIA2
99	IB	179	IIB3	216	IIB1	252	IA
128	IIC2	180	IA	217	IIA1	254	IA
129	IIA1	184	IIB3	218	IIB2	255	IIA1
130	IIB2	186	IB	219	IIB2	256	IA
137	IID2	187	IIB3	222	IIB1	257	IA
138	IA	188	IIA2	223	IIB1	258	IA
141	IIA1	189	IIB1	225	IIB2	259	IIA2
143	IIA1	190	IIB1	226	IIA1	260	IA
147	IB	191	IID1	228	IID1	261	IIB2
148	IA	192	IIA2	229	IIA2	262	IIA1
150	IB	194	IC	230	IIA2	264	IIA2
151	IB	195	IIB1	231	IIB1	266	IIA2

word "abortion," as herein used, means a fetus delivered dead, or non-viable, even though in some cases the period of gestation was approximately normal. This stand was taken, as the end result in the field is similar and to save confusion in tabulating. Results are recorded in those cases where cultural findings were made of aborted fetuses. Udder infection, if listed as positive, means that *Brucella abortus* was isolated from the milk of the animal during life, or from the udder at slaughter.

Summary of Blood Serum Agglutination Tests

I. Sixteen animals gave positive reactions in 1:25 but were never positive in 1:50.

A. Seven (44%) of these animals (cows 188, 192, 199, 229, 245, 251 and 266) were finally classed as negative.

B. Nine (56%) of these animals (cows 71, 83, 143, 153, 194, 210, 226, 255 and 262) were finally classed as suspicious.

II. Eighteen animals gave positive reactions in 1:50.

A. Thirteen were positive in 1:50 from one to five months.

1. Seven (54%) of these animals (cows 47, 214, 218, 219, 230, 240 and 259) were finally classed as negative.

2. Three (23%) (cows 49, 202 and 217) were finally classed as suspicious.

Table II—*Outline of final grouping according to agglutination reactions*

Grouping	Animals	See Tables
I. Constant in blood reaction:		
A. Negative throughout period of tests	15	III, IV
B. Positive throughout period of tests	11	V, VI
C. Suspicious throughout period of tests	2	VII, XIII
II. Changed in blood reaction:		
A. Originally negative, became suspicious and		
1. Remained suspicious	13	IX, X
2. Became negative	13	XI, XII
3. Became positive	6	XIII, XIV
B. Originally negative, became positive and		
1. Remained positive	15	XV, XVI
2. Became negative	7	XVII, XVIII
3. Became suspicious	6	XIX, XX
C. Originally suspicious,		
1. Became negative	4	XXI, XXII
2. Became positive	3	XXIII, XXIV
D. Originally positive,		
1. Became suspicious	4	XXV, XXVI
2. Became negative–positive–suspicious	1	XXVII, XXVIII
Total	100	

TABLE III—*Group IA. Negative history throughout*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
41	31	6	273			
			273		Died 9-27-28	
			269			
			269	Dead at delivery. Retained fetal membranes. Streptococcus isolated	Septicemia following last parturition	Negative
			264	Twins. Dead at delivery		
			267	Dead at delivery		
138	48	6	275			
			271			
			272	Dead at delivery	Living	Negative
			233	Aborted. *B. coli* isolated		
			279	Retained fetal membranes		
			287			
148	27	4	278			
			283		Slaughtered 6-7-28	Negative
			280	Retained fetal membranes		
			272	Retained fetal membranes		
164	28	5	280			
			281			
			284		Slaughtered 6-8-28	Negative
			284			
			289			
173	43	5	259	Dead at delivery		
			275			
			266	Dead at delivery	Living. Pregnant	Negative
			269			
			277	Retained fetal membranes		

TABLE III—*Group IA. Negative history throughout—Concluded*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
180	43	4	290 294 283 288		Sold. Pregnant	Negative
196	43	2	285 302		Sold. Pregnant	Negative
243	35	2	277 276		Living. Pregnant	Negative
245	35	1	291		Living. Pregnant	Negative
252	35	1	280	Became sterile, cystic ovaries	Slaughtered 2-28-30	Negative
254	27	2	287 285		Living	Negative
256	28	1	—	Normal calf	Living. Pregnant	Negative
257	29	1	270		Living. Pregnant	Negative
258	28	1	284		Living . Pregnant	Negative
260	28	2	278 279		Living	Negative

TABLE IV—*Agglutination reactions of group IA*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
41	1926												
	1927												
	1928										Dead		
138	1926												
	1927				P								
	1928												
	1929						T						
	1930												
148	1926												
	1927					T		P	T		T	T	T
	1928							Dead					
164	1926												
	1927								T				
	1928							Dead					
173	1926												
	1927		P										
	1928												
	1929												
	1930												
180	1926												
	1927				T		P	T			T	T	
	1928												
	1929								T		Sold		
196	1926												
	1927				P	T	T						
	1928	T		T	T				T	T			T
	1929		T					T	T		Sold		
243	1927										P		
	1928												
	1929												
	1930												

TABLE IV—*Agglutination reactions of group IA—Concluded*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
245	1927	– – – – –	– – – – –	– – – – –	+ – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	P – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
252	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
254	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –		– – – – –	– – – – –
	1930		– – – – –										
256	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	
	1930	– – – – –	– – – – –										
257	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
258	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
260	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										

(2896)

TABLE V—*Group IB. Positive history throughout*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
42	26	7	132	Aborted		
			271	Dead at delivery	Slaughtered 4-5-28	
			262	Twins. Dead at delivery. Retained fetal membranes		Negative
			260	Dead at delivery	Bad udder	
			275	Retained fetal membranes		
			268	Dead at delivery		
44	38	8	—	Normal calf		
			193	Aborted		
			279		Slaughtered 2-19-30	
			274			
			277	Retained fetal membranes	Chronic streptococcic mastitis.	Positive
			267			
			267		Sterile	
			—	Normal calf. Treated 10 mos. for sterility after last parturition		
95	31	6	240	Twins. Aborted		
			290			
			290		Slaughtered 9-20-28	
			102	Aborted		Positive
			160	Aborted. Retained fetal membranes		
			112	Aborted. Treated one year for sterility after last parturition	Sterile	

TABLE V—*Group IB. Positive history throughout—Continued*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
99	43	5	270			
			81	Aborted		
			136	Aborted. Retained fetal membranes. *Br. abortus* isolated	Slaughtered 9-18-29	
			286			Positive
			277	Retained fetal membranes. Treated for sterility over a period of 17 mos. after last parturition	Sterile	Positive
147	34	6	239	Aborted		
			155	Aborted	Died 1-2-29	
			274			Negative
			280		Chronic indurative pneumonia	
			280			
			275			
150	48	6	220	Aborted		
			275		Living. Now under treatment for sterility	Positive
			274			
			275			
			276			
			275			
151	48	6	266	Dead at delivery		
			259	Dead at delivery		
			256		Living. Pregnant	Positive
			211	Aborted		
			273			
			279			

TABLE V—*Group IB. Positive history throughout—Concluded*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
162	48	7	282 279 285 282 287 274 282	Died two days old	Living. Pregnant	Positive
186	30	2	268 155	Aborted. Sterile for one year	Slaughtered 9-27-28 Sterile	Positive
198	47	4	279 274 242 288	Retained fetal membranes Aborted. Died 4 hours after birth	Living	Positive
211	22	0		Permanently sterile. Treated for sterility. Served 13 times over a period of 17 mos.	Sterile	Positive

TABLE VI—*Agglutination reactions of group IB*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
42	1926			+ + + + −	+ + + + +	+ + + + +	+ + + + P	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + P	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1928	+ + + P −	+ + + + +	+ + + P T	+ + + P T	Dead							
44	1927	+ + + + −	+ + + P −	+ + + P P	+ + + + +	+ + + P T	+ + + + +	+ + + + P	+ + + + +	+ + + + P	+ + P P P	+ + + + +	+ + + P P
	1928	+ + + + +	+ + + + +	+ + + P T	+ + + + +	+ + + + +	+ + + + P	+ + + + T	+ + + + P	+ + + + P	+ + + + T	+ + + + P	+ + + + P
	1929	+ + + + P	+ + + + P	+ + + + +	+ + + + +	+ + + + +	+ + P − −	+ + + + +	+ + + + +	+ + + P T	+ + + + +	+ + + + +	+ + + + +
	1930	+ + + + +	+ + + + +	Dead									
95	1926			+ + + + +	+ + + + +	+ + + + +	+ − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + P T	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	Dead		
99	1926			+ + + + P	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + −	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + P	+ + + + P	+ + + + P	+ + + + +	+ + + + P	+ + + + P	+ + + + T
	1928	+ + + + +	+ + + + +	+ + P T −	+ + + + +	+ + P T −	+ + + P T	T − − − −	+ + + P −	+ + T − −	+ P T − −	+ + P T −	+ + + − −
	1929	+ + + P T	+ + + P T	+ + P T −	+ + + P T	+ + P − −	+ + P T −	+ + + + +	+ + + + +	+ + + + −	Dead		
147	1926			P P + + P	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + − −	+ + + + +	+ + + − −	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + P P	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + P	+ + + + P	+ + + + +
	1929	Dead											
150	1926			+ + + + +	+ + + + P	+ + + + P	+ + + + +	+ P − − −	+ + + + +	+ + + + +	+ + + − −	+ + + + P	+ + + P P
	1927	+ + + + −	+ + − − −	− − − − −	+ + + − −	+ P T − −	+ P T − −	+ P P T −	+ P − − −	+ P T − −	+ + + P P	+ + P + +	+ + + P P
	1928	+ + + + P	+ + P T −	+ + P T −	+ + + T −	+ + P − −	+ + + P −	+ + + T −	+ + + P T	+ + + + +	+ + + P T	+ + + + +	+ + + + +
	1929	+ + + + P	+ + P T −	+ + P T −	+ + + − −	+ + T − −	+ + P T −	+ + + P T	+ + + + +	+ + + + +	+ + + P −	+ + + P −	+ + P P T
	1930	+ + P T −	+ + P − −										

TABLE VI—*Agglutination reactions of group IB—Concluded*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
151	1926			+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + P	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1929	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + P	+ + + + +
	1930	+ + + + +	+ + + + +										
162	1926			+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + P T	+ + + + +	+ + + + P	+ + + + +	+ + + + +	+ + + + P	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + P T	+ + + + P	+ + + + P	+ + + + +	+ + + + +	+ + + + +	+ + + + P	+ + + + P T
	1929	+ + + + +	+ + + + +	+ + + + T	+ + + + +	+ + + + +	+ + + P T	+ + + + +	+ + + + +	+ + + P T	+ + + + +	+ + + + +	T P + + P
	1930	+ + P T −	P P T − −										
186	1926			+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +		+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + T	+ + + + T	+ + + + T	+ + + T −	+ + + P T	+ + + + +	+ + P T −	Dead		
198	1926			+ + + − −	+ + P − −	+ + P − −	+ + − − −	+ + − − −	+ + + + +	+ + + − −	+ + P − −	+ + + − −	+ + + + −
	1927	+ P − − −	+ P − − −	+ + + + P	+ + + + +	+ + + P T	+ + + + +	Hem.	+ + T T T	+ + + + T	+ + T − −	+ + + + +	+ + P P P
	1928	+ + + + +	+ + + + +	+ + + P T	+ + + + P	+ + + + P	+ + + + +	+ + + + P	+ + + + +	+ P T − −	+ + + P T	+ + + + P	+ + + + +
	1929	+ + + + +	− − − − −	+ + + + +	+ + + + +	+ + + + +	− − − − −	+ + + + +	P T − − −	+ + + + +		+ + + + +	+ + + + +
	1930	+ + + P T	+ + + P T										
211	1926			+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ P P − −	+ P P − −	P P − − −	P − − − −	+ + P − −	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + T	+ + + T −	+ + P T −	+ + + + +	+ + − − −	+ + + P −	+ P − − −	+ + + + P
	1928	Dead											

TABLE VII—*Group 1C. Suspicious history throughout*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
49	46	6	276	Normal calf	Slaughtered 12-5-29	Negative
			271			
			254			
			268	Died at 4 days of pneumonia	Sterile	
			275	Treated for sterility over a year		
194	48	4	280		Living. Pregnant	Negative
			274			
			272			
			278			

TABLE VIII—*Agglutination reactions of group 1C*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
49	1926			– – – – –	– – – – –	– – – – –	– – – – –	P – – – –	P – – – –	+P – – –	+P T – –	+P T – –	P T – – –
	1927	+P – – –	+P T – –	P – – – –	+ +P – –	P T – – –	+T – – –	+P T – –	+ +T – –	+P T – –	P T – – –	P T – – –	P T – – –
	1928	+P T – –	+P T – –	P T – – –	+ +– – –	P T – – –	+P T – –	+P T – –	+ +P T –	+ +P T –	+P T T –	+P T – –	+P T – –
	1929	+P T – –	+ +P – –	+ +T – –	+P T – –	+P T – –	+P T – –	+P T – –	+ + +P –	+P T – –	+ +P – –	+P T – –	+P T – –
	1930	Dead											
194	1926			+P – – –	+P – – –	P – – – –	– – – – –	P – – – –	+P – – –	P P – – –	– – – – –	T – – – –	P P – – –
	1927	– – – – –	+P – – –	– – – – –	+ – – – –	+T – – –	P – – – –	+T – – –	T – – – –	P T – – –	P – – – –	P T – – –	T – – – –
	1928	P T – – –	P T – – –	+T – – –	+T – – –	+P – – –	P T – – –	+ – – – –	+T – – –	P T – – –	T – – – –	P T – – –	– – – – –
	1929	+P – – –	P T T – –	P T – – –	P T – – –	P T – – –	T – – – –	P T – – –	+P T T –	T – – – –	T – – – –	– – – – –	+P T – –
	1930	T – – – –	P T – – –										

TABLE IX—*Group IIA1. Originally negative, became suspicious and remained suspicious*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
71	48	8	284 283 277 281 279 282 282 282		Living. Pregnant	Negative
83	43	10	287 — 285 280 285 287 293 297 292 288	Normal calf	Slaughtered 9-18-29 Sterile	Negative
129	42	8	274 276 273 274 279 276 276 280		Slaughtered 8-19-29 Poor type	Negative

TABLE IX—*Group IIA1. Originally negative, became suspicious and remained suspicious—Continued*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
141	48	5	280			
			216	Aborted		
			278		Living.	Negative
			252	Aborted twins. Retained fetal membranes. Streptococcus isolated		
			281	Under treatment for sterility. 20 mos. since last parturition	Sterile	
143	46	5	281			
			283			
			277		Slaughtered 12-4-29 Unprofitable	Negative
			278			
			276			
153	45	6	276			
			273			
			274		Slaughtered 11-12-2(	Negative
			276			
			276			
			277			
202	48	4	266			
			275			
			275		Living. Pregnant	Negative
			276	Retained fetal membranes		
210	25	1	283	Sterile over a year of treatment	Slaughtered. Sterile	Positive

TABLE IX—*Group IIA1. Originally negative, became suspicious and remained suspicious—Concluded*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
217	39	2	283 275		Slaughtered 5-24-29 Sterile	Positive
226	40	3	274 273 147	*Aborted twins. Development anomaly of fetal membranes	Living	Negative
250	29	1	272		Living. Pregnant	Negative
255	29	2	277 286		Living	Negative
262	24	0		Served 7 times over a period of 13 months	Slaughtered 11-4-29 Sterile	Negative

*The fetal membranes had only 17 placental attachments.

TABLE X—*Agglutination reactions of group IIA1*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
71	1926												
	1927				P	T			T				
	1928		P T	T	P T						P T	T	+T
	1929	P	P T			T		P T	P T	P T	T	T	T
	1930	T											
83	1926						+		+	P P			
	1927			P		P T		P	+T	T			T
	1928		T	T	+P T	T			P T	T		T	T
	1929	T	P T	T	+			P T	+P T	T T	Dead		
129	1926												
	1927					T							
	1928		P T		P T					T	T		T
	1929	T	P T	P T		T		P T	P T	Dead			
141	1926												
	1927				P				T				
	1928				T								T
	1929	T	T	T				T	T		P T	T	P P T
	1930	P P T	T										
143	1926												
	1927				P			P T	+T		P		
	1928		T		T								
	1929			T				T	P T		T		T
	1930	Dead											
153	1926												
	1927												
	1928		P T	P		T						P	+P
	1929	P T	T	T		T		+P T	+P T	P T	T	P	Dead
202	1926								+ +P				
	1927					T							T
	1928		T	P T		T				T			
	1929								T		P T		
	1930												

TABLE X—*Agglutination reactions of group IIA1—Concluded*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
210	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1927	– – – – –	– – – – –	– – – – –	+ – – – –	T – – – –	– – – – –	T – – – –	– – – – –	– – – – –	T – – – –	– – – – –	T – – – –
	1928	– – – – –	– – – – –	P T – – –	Dead								
217	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	P – – – –	P – – – –	– – – – –	– – – – –
	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –
	1928	– – – – –	+T – – –	+ +T – –	+P – – –	+T – – –	+P – – –	+P T – –	T – – – –	T – – – –	T – – – –	+T – – –	+T – – –
	1929	+T – – –	+P – – –	+T – – –	P T – – –	+T – – –	Dead						
226	1926						– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	+P – – –	– – – – –
	1928	– – – – –	T – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	P T – – –	– – – – –	– – – – –	– – – – –	P T – – –
	1930	T – – – –	– – – – –										
250	1927									– – – – –		– – – –	
	1928	– – – – –	– – – – –	P – – – –	T – – – –	– – – – –	– – – – –	– – – – –	T – – – –	T – – – –	P T – – –	T – – – –	P T – – –
	1929	P – – – –	T – – – –	T – – – –	T – – – –	P – – – –	T – – – –	P T – – –	P T – – –	– – – – –	T – – – –	P T – – –	– – – – –
	1930	T – – – –	– – – – –										
255	1927										– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	T – – – –	T – – – –	T – – – –	T – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	P T – – –	T – – – –	P T – – –	P T – – –	T – – – –	P T – – –	+P T – –	– – – – –	T – – – –	T – – – –	T – – – –
	1930	P T – – –	P T – – –										
262	1927											– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	+P T – –	– – – – –	– – – – –	– – – – –	Dead	

TABLE XI—*Group IIA2. Originally negative, became suspicious and then became negative*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
73	48	8	278 274 279 220 135 273 279 261	Aborted. Retained fetal membranes Aborted	Living. Pregnant	Negative
152	48	6	296 274 274 269 272 280	Dead at delivery	Living. Pregnant	
155	48	6	273 275 275 280 281 278		Living. Pregnant	Negative
188	48	4	273 278 277 280	Retained fetal membranes	Living. Pregnant	Negative
192	48	4	283 280* 285 280		Living. Pregnant	Positive

TABLE XI—*Group IIA2. Originally negative, became suspicious and then became negative—Concluded*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
199	48	4	257 274 276 278	Dead at delivery. Cow was thrown heavily a few days previously when bled Dead at delivery	Living. Pregnant	Negative
229	40	2	273 273		Living. Pregnant	Negative
230	40	3	266 270 249	Retained fetal membranes	Living. Pregnant	Negative
241	36	2	288 164	Aborted mummified fetus	Living	Negative
251	32	2	277 178	Aborted. Isolated *Vibrio fetus*	Living	Negative
259	28	1	272		Living. Pregnant	Negative
264	25				Living. Pregnant	Negative
266	21			Sterile	Living	Never cultured

**Br. abortus* was isolated from milk in one instance only, in 1927.

TABLE XII—*Agglutination reactions of group IIA2*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
73	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	P – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1927	– – – – –	– – – – –	– – – – –	P – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	P T – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
152	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1927	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	P T – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
155	1926			– – – – –	– – – – –	– – – – –	– – – – –	P – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1927	– – – – –	– – – – –	– – – – –	P – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –
	1928	P T – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	T – – – –	– – – – –	T – – – –	– – – – –	– – – – –	P T – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
188	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	T – – – –	– – – – –
	1927	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	T – – – –	P – – – –	+T – – –	– – – – –	T – – – –	T – – – –	– – – – –
	1928	T – – – –	– – – – –	– – – – –	– – – – –	T – – – –	T – – – –	– – – – –	T – – – –	T – – – –	– – – – –	T – – – –	– – – – –
	1929	P – – – –	P T – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
192	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1927	– – – – –	– – – – –	+P – – –	P – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
199	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	P P – – –	– – – – –	– – – – –	– – – – –	P T – – –
	1927	P – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	T – – – –	T – – – –	T – – – –	P – – – –	– – – – –
	1928	T – – – –	T – – – –	P T – – –	– – – – –	T – – – –	T – – – –	– – – – –	T – – – –	P – – – –	– – – – –	P – – – –	T – – – –
	1929	P T – – –	P T – – –	+P – – –	T – – – –	T – – – –	– – – – –	P T – – –	P T – – –	T – – – –	T – – – –	– – – – –	– – – – –
	1930	T – – – –	– – – – –										

TABLE XII—*Agglutination reactions of group IIA²—Concluded*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
229	1926												
	1927				+P	P T	P	+T	+T	P T	P		
	1928		P T	P T	T	T		P	P T	P T			P T
	1929	T	P T	P T	P	P T		P T	P T				
	1930		T										
230	1926								+ + P P	+	T T		
	1927				+P	T		P	T	T			
	1928				T					T			
	1929								T				
	1930												
241	1927												P
	1928		P T	P									
	1929												
	1930												
251	1927												
	1928			+T									
	1929								P T				T
	1930												
259	1927												
	1928				+P	+ + T							
	1929								T				
	1930												
264	1928								T				
	1929					T		P T	T				
	1930												
266	1928												
	1929								+T				
	1930												

TABLE XIII—*Group IIA3. Originally negative, became suspicious and then positive*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
89	34	4	265 280 293 280	Retained fetal membranes. Became sterile	Sold for experiment purposes, 10-28	Negative
174	43	4	274 279 275 282	Dead at delivery	Slaughtered 1-8-30 Pregnant. Poor producer	Negative
232	48	4	219 273 280 281	Aborted. *Br. abortus* isolated	Living	Negative
236	25	0		Bred but once. Treated for sterility one year. Never conceived. *Br. abortus* isolated from uterus and udder	Slaughtered 9-27-28 Sterile	Positive
237	25			Bred 8 times. Treated for sterility over a year. Never conceived. *Br. abortus* isolated from udder	Slaughtered 10-5-28 Sterile	Positive
238	39	2	208 274	Aborted Cystic ovaries. Under treatment 1 year	Living. Sterile	Positive

TABLE XIV—*Agglutination reactions of group IIA3*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
89	1926												
	1927	P	P T		+P	T	P T	P T	+P T	+T	P T	P T	T
	1928		T	T	+P T	T		P T	+ + +T	+ +P T	+P	T	T T
174	1926												
	1927				P	T		+P	T	T	T	T	T
	1928	+P T	P T	P T	T	+ +P	+ + + +P	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + +P
	1929	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + +P T	+ + +P T	+ + +P T	+ + + +T	+ + + +P	+ + + +P	+ + + + +
	1930	+ + + + +	Dead										
232	1926					+P							+ +P
	1927		+ + + +	+ + + + +	+ + + + +	+ + + +P	+ + +P T	+P P	+P T	+ +T	+ +P T	+ +P T	+ +P T
	1928	+P T	+ + +P T	+ + +	+P T	+P	T		T	P T		P T	
	1929	P P T	P P P	P T	P P P T	P P T		P T	T			T	
	1930	+P T											
236	1926												
	1927												
	1928		P	+ +P	+ +P T	+ + + +P	+ + + + +	+ + + + +	+ + + + +	+ + + + +	Dead		
237	1926											+ +P	+ +
	1927	+ +P	+ +	+ + +P	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + +P
	1928	+ + + + +	+ + + + +	+ + + +	+ + +P P	+ + + + +	+ + + + +	+ + + +P	+ + + + +	+ + + +P	Dead		
238	1926												
	1927						T	P T	+ + + +T	+ + + + +	+ + +P P	+ + + +P	+ + + +P
	1928	+P T	+ + + + +	+ + + + +	+P P T T	+ + + +P	+ + +P T	+ +P P T	+ + + +P	+ +P T T	+ + +P P	+ + +P T	+ + + +P
	1929	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+P T	+ + + + +	+ +P T T	+ + +P T	+ +P T	+ +P T	+ + + + +
	1930	+ + + +P	+ +P T										

TABLE XV—*Group IIBI. Originally negative, became positive and remained positive*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
64	22	7	280 276 281 293 283 272 274		Died 12-5-27. Posterior paralysis	Negative
166	38	2	293 216	Aborted. Treated for sterility over a year	Slaughtered 4-17-29 Sterile	Positive
168	47	5	274 271 282 272 285	Retained fetal membranes	Living	Positive
189	39	3	283 288 289		Slaughtered 11-12-29 Sterile	Positive
190	31	1	288	At slaughter found a mummified fetus	Slaughtered 9-26-28	Negative
195	28	3	268 208 257	Aborted Dead at delivery. Retained fetal membranes. *Br. abortus* isolated from fetus	Died 6-27-28. Septicemia	Positive
203	22	1	233	Aborted. *Br. abortus* isolated from fetus and fetal membranes. Became sterile	Slaughtered 12-12-27 Sterile	Positive

TABLE XV—*Group IIB1. Originally negative, became positive and remained positive—Concluded*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
204	45	3	178 293 292	Aborted	Slaughtered 11-4-29 Unprofitable	Positive
208	22	2	277 217	Aborted. Became sterile	Slaughtered. TB and sterile	Positive
215	25	0		Never became pregnant. Served 12 times. Treated for 12 mos.	Slaughtered. Sterile	Negative
216	47	3	167 285 289	Aborted. *Br. abortus* isolated	Living. Sterile	Positive
222	30	1	253	Twins aborted. *Br. abortus* isolated. Became sterile	Slaughtered 10-5-28 Sterile	Negative
223	35	1	243	Aborted	Slaughtered 4-24-29 Pregnant	Positive
231	37	1	264	Dead at delivery. *Br. abortus* isolated. Became sterile	Slaughtered 6-19-29 Sterile	Positive
233	41	2	280 283	Treated 5 mos. for sterility Became sterile	Slaughtered 2-25-30 Sterile	Positive

TABLE XVI—*Agglutination reactions of group IIB1*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
64	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	T − − − −	− − − − −	+P − − −
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + +P	+ + + + +	+ + +P T	+ + + + −	+ + + +T	+P P P P	+ + +P P	+ + + + +
	1928	Dead											
166	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1929	+ + + +P	+ + + +P	+ + + +P	+ + +P	Dead					+ + + +P	+ + + + +	+ + + +P
168	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1929	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + +P
	1930	+ + + + +	+ + + + +							++P T −	+ + + + +	+ + + + +	+ + + + +
189	1926								− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1927	− − − − −	− − − − −	− − − − −	P − − − −	− − − − −	− − − − −	− − − − −	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +		+ + + + +	+ + + + +	+ + + + +
	1929	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + +P	+ + + + +	+ + + + +	++P − −	+ + + + +	+ + + + +	Dead
190	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1927	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	Dead		
195	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	P − − − −	− − − − −	− − − − −	T − − − −	− − − − −
	1927	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	+ + + +T	+ + + + +	+ + + + +	+ + + + +	+ + +P T
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	Dead					
203	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1927	+ − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	P + + + +	+ + + + +	+ + +P T	+ + + + +
	1928	Dead											
204	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	P T − − −	+ + + +P
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + +P	+ + +P T	+ + + + +	+ + + + +	+ +P P T	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + +P P	+ + + + +	+ + + +P	+ + + + +
	1929	+ + + + +	− − − − −	− − − − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	Dead

TABLE XVI—*Agglutination reactions of group IIB1—Concluded*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
208	1926												
	1927	Dead											
215	1926												
	1927				Dead								
	1928												
216	1926												
	1927												
	1928												
	1929												
	1930												
222	1926												
	1927											Dead	
	1928												
223	1926												
	1927												
	1928												
	1929					Dead							
231	1926												
	1927												
	1928							Dead					
	1929												
233	1926												
	1927												
	1928												
	1929			Dead									
	1930												

TABLE XVII—*Group IIB2. Originally negative, became positive and then negative*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
130	26	5	280 260 262 275 286	Dead at delivery Retained fetal membranes	Slaughtered 4-9-28. Pregnant. Unprofitable	Negative
214	48	2	282 275	Retained fetal membranes	Living. Pregnant	Negative
218	48	3	277 277 275		Living, Pregnant	Negative
219	48	4	278 273 272 276		Living	Negative
225	42	3	274 279 275		Living. Pregnant	Negative
235	41	3	281 281 281		Living	Negative
261	28	1	—	Calf normal	Living	Negative

TABLE XVIII—*Agglutination reactions of group IIB2*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
130	1926			− − − − −	− − − − −	− − − − −	+ + + + +	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1927	− − − − −	− − − − −	− − − − −	P − − − −	T − − − −	− − − − −	− − − − −	T − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1928	− − − − −	T − − − −	− − − − −	P T − − −								
214	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	+ + + P T
	1927	+ + + − −	+ + + − −	− − − − −	+ + + + −	+ + + + +	+ + P T −	+ + T − −	+ + P T −	+ + P T −	+ − − − −	+ P − − −	T T − − −
	1928	P T − − −	P T − − −	+ + + T −	P T − − −	P T − − −	− − − − −	P T − − −	P T − − −	P T − − −	T − − − −	− − − − −	T − − − −
	1929	T − − − −	T − − − −	− − − − −	T − − − −	− − − − −	T − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1930	T − − − −	− − − − −										
218	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	+ − − − −	+ + P T −	+ + + − −
	1927	+ + − − −	+ P − − −	P − − − −	− − − − −	P P T − −	+ − − − −	+ P − − −	− − − − −	+ − − − −	+ + P − −	+ T − − −	− − − − −
	1928	− − − − −	T − − − −	T − − − −	T − − − −	− − − − −	− − − − −	P T − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1929	− − − − −	− − − − −	− − − − −	− − − − −	T − − − −	− − − − −	− − − − −	P T − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1930	− − − − −	− − − − −										
219	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1927	+ + − − −	P T − − −	T − − − −	+ − − − −	P T − − −	P − − − −	T − − − −	T − − − −	T − − − −	T − − − −	− − − − −	− − − − −
	1928	− − − − −	− − − − −	+ T − − −	P T − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	T − − − −	− − − − −
	1929	P T − − −	T − − − −	T − − − −	T − − − −	T − − − −	T − − − −	P T − − −	P T − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1930	− − − − −	− − − − −										
225	1926							− − − − −				− − − − −	− − − − −
	1927	− − − − −	− − − − −	− − − − −	− − − − −	P T − − −	P − − − −	+ P T − −	+ T − − −	+ T − − −	P T − − −	+ + + + +	T − − − −
	1928	− − − − −	− − − − −	− − − − −	P T − − −	T − − − −	P T − − −	− − − − −	P T − − −	P − − − −	T − − − −	− − − − −	− − − − −
	1929	T − − − −	T − − − −	− − − − −	− − − − −	P T − − −	− − − − −	P T − − −	P T − − −	− − − − −	T − − − −	T − − − −	− − − − −
	1930	− − − − −	− − − − −										
235	1926											− − − − −	− − − − −
	1927	− − − − −	+ + + + −	+ + + − −	+ + P − −	+ P T − −	+ − − − −	+ − − − −	+ T − − −	− − − − −	T − − − −	T − − − −	T − − − −
	1928	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	T − − − −	− − − − −	− − − − −	− − − − −
	1929	− − − − −	− − − − −	− − − − −	− − − − −	T − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1930	− − − − −	− − − − −										
261	1927											− − − − −	− − − − −
	1928	− − − − −	+ + + P T	+ + + P T	+ + P − −	+ T − − −	P T − − −	+ P T − −	+ + T − −	+ T − − −	T − − − −	P − − − −	T − − − −
	1929	− − − − −	P T − − −	− − − − −	− − − − −	T − − − −	T − − − −	T − − − −	P T − − −	T − − − −	− − − − −	− − − − −	T − − − −
	1930	T − − − −	T − − − −										

TABLE XIX—*Group IIB3. Originally negative, became positive and then suspicious*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
159	34	4	279 269 279 272	*Br. abortus* isolated from fetal membranes Twins. Retained fetal membranes. Septicemia	Died 1-1-29. Septicemia	Negative
167	34	0		Bred 1 yr. Treated for sterility. Never conceived	Sold for experiment purposes 10-1-28	Negative
179	43	4	291 284 294 292	 Retained fetal membranes	Living	Negative
184	45	4	282 292 290 291	 Retained fetal membranes Retained fetal membranes	Slaughtered 11-12-29 Unprofitable	Negative
187	48	5	270 275 222 279 283	 Aborted. Retained fetal membranes	Living	Negative
209	43	4	229 280 241 251	Aborted. *Br. abortus* isolated Aborted. Retained fetal membranes Delivered twins. One dead, one lived. Retained fetal membranes	Slaughtered 9-19-29 Sterile	Negative

TABLE XX—*Agglutination reactions of Group IIB3*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
159	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1927	+ + + + +	+ + + + +	+ + + P −	+ + + + +	+ + P T −	+ + + P −	+ + P T −	+ + + − −	+ + T − −	+ + + − −	+ + P − −	+ P T − −
	1928	+ + + P T	+ + P T −	+ + + P −	+ + + T −	+ + P − −	+ P T − −	P P T − −	+ + P − −	+ + T − −	+ + P − −	+ + P T −	+ + T − −
	1929	Dead											
167	1926			− − − − −	− − − − −	− − − − −	+ + + − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + + P	+ + + + +	+ + + + + P	+ + + P T	+ + + P P	+ + + P −	+ + + + +	+ + + P T	+ + + + P	+ + + P T
	1928	+ + + + +	+ + + T −	+ + + + P	+ + + + +	+ + + P −	+ + + P −	T − − − −	+ P P T −	T − − − −	− − − − −	− − − − −	P T − − −
	1929	Sold											
179	1926								− − − − −	P − − − −	P − − − −	+ + + + T	+ + + + T
	1927	+ + + P −	+ + + P T	+ + + + −	+ + + + P	+ + + + T	+ + + P T	+ − − − −	+ + + P −	+ + + − −	+ P T − −	+ + + − −	+ + P − −
	1928	+ + P T −	+ + P T −	+ + + T −	+ + P − −	+ + P T −	+ P − − −	+ + P − −	+ + P − −	+ + T − −	+ P T − −	+ P − − −	+ P − − −
	1929	T − − − −	T − − − −	P T − − −	+ T − − −	− − − − −	+ T − − −	+ P T − −	+ + P T −	P − − − −	P T − − −	P T − − −	P T − − −
	1930	P P T − −	P T − − −										
184	1926			− − − − −	− − − − −	− − − − −	− − − − −	+ − − − −	+ + + + −	+ + + + +	+ + + + +	+ + + + −	+ + + + T
	1927	+ + + − −	+ + P − −	+ + − − −	+ + P − −	+ P T − −	+ P T − −	+ P − − −	+ T − − −	+ T − − −	+ T − − −	+ P T − −	+ − − − −
	1928	+ + P T −	+ P T − −	+ + + P −	+ + + P −	+ + + P −	+ P − − −	+ P − − −	+ P − − −	+ P T − −	+ P − − −	+ T − − −	+ T − − −
	1929	+ P T − −	+ + T − −	+ + P − −	+ P T − −	+ + T − −	+ P T − −	P T − − −	+ + P T −	− − − − −	P P T − −	P T − − −	Dead
187	1926			− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1927	T − − − −	T − − − −	T − − − −	T − − − −	T − − − −	T − − − −	− − − − −	T − − − −	− − − − −	− − − − −	− − − − −	+ + + + +
	1928	+ + + + +	+ + + + +	+ + + P −	+ + + + T	+ + + + T	+ P − − −	+ + P T −	+ + P − −	+ + T − −	+ + P − −	+ + P T −	+ + T − −
	1929	+ P T − −	+ + T T −		+ P T − −	+ + T − −	T − − − −	+ P T − −	+ + P T −	P T − − −	+ P T − −	P T − − −	+ + P T −
	1930	+ P T − −	P T − − −										
209	1926			− − − − −	− − − − −	+ + + P −	+ + + P −	+ + + + +	+ + + + +	+ + + + +	+ + P − −	+ + + + −	+ + + P −
	1927	+ + + − −	+ + + − −	+ + − − −	+ + + − −	+ + P T −	+ + − − −	+ P − − −	+ + P − −	+ P − − −	+ + P T −	+ + P − −	+ + + − −
	1928	+ + + + T	+ + P T −	+ + + T −	+ + + − −	+ + T − −	+ + T − −	+ + T − −	+ + T − −	+ + P − −	+ + T − −	+ + P T −	+ + P − −
	1929	+ + + P T	+ + + P −	+ P − − −	+ P T − −	+ P − − −	T − − − −	+ P T − −	+ + P T −	P T − − −	Dead		

TABLE XXI—*Group IIC1. Originally suspicious and became negative*

ANIMAL	AGGLUITNATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
47	35	7	265	Dead at delivery		
			—	Normal calf		
			272	Twins. Retained fetal membranes	Slaughtered 1-11-29	
			279		Unproductive.	Negative
			271		Poor type	
			280			
			278			
62	39	7	205	Aborted		
			287			
			281		Slaughtered 5-31-29	
			278		Poor producer.	Negative
			278		Sterile	
			283			
			277	Twins. One delivered dead. One died at two days. Became sterile		
240	36	2	271		Living	Negative
			280			
242	35	2	289		Living	Negative
			290			

TABLE XXII—*Agglutination reactions of group IIC1*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
47	1926			– – – – –	– – – – –	– – – – –	– – – – –	+P – – –	++ – – –	– – – – –	T – – – –	T – – – –	– – – – –
	1927	P – – – –	– – – – –	– – – – –	+P T – –	+T – – –	+ – – – –	+ – – – –	+ – – – –	+ – – – –	P – – – –	P – – – –	T – – – –
	1928	P T – – –	– – – – –	T – – – –	P T – – –	– – – – –	– – – – –	– – – – –	T – – – –	T – – – –	– – – – –	– – – – –	T – – – –
	1929	T – – – –	Dead										
62	1926			– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1927	– – – – –	P – – – –	– – – – –	+ – – – –	T – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	– – – – –	– – – – –	P T – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	T – – – –	T – – – –	T – – – –	T – – – –	– – – – –	Dead						
240	1927			+P – – –	++ – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1928	– – – – –	P T – – –	P – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	T – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	– – – – –	– – – – –										
242	1927				+P – – –	T – – – –	– – – – –	T – – – –	T – – – –	– – – – –	– – – – –	P T – – –	– – – – –
	1928	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1929	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	– – – – –	T – – – –	– – – – –	– – – – –	– – – – –	– – – – –
	1930	T – – – –	– – – – –										

TABLE XXIII—*Group IIC2. Originally suspicious and became positive*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
19	48	10	278			
			211	Aborted		
			277			
			283			
			276		Living	Negative
			271			
			269			
			266			
			269			
			268	Treated for sterility 8 mos.		
128	48	8	295			
			274			
			252	Aborted		
			277		Living. Pregnant	Negative
			270			
			214	Aborted		
			279			
			272			
205	45	3	257	Died 12 hrs. Retained fetal membranes	Slaughtered 12-10-29	Positive
			259	Died 2 days. *B. coli* isolated	Unprofitable	
			284			

TABLE XXIV—*Agglutination reactions of group IIC2*

										SEPT.	OCT.	NOV.	DEC.

TABLE XXIV—*Agglutination reactions of group IIC2*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
19	1926			++P −−	++P T −	++P −−	++P −−	+++P −	++−−−	+++P −	++−−−	+++P −	+++P −
	1927	+++−−	+++P T	++T −−	+P T −−	+++T −	+++P T	++−−−	++P T T	++P T T	++P P T	++P P −	++++T
	1928	++++P	+++++P	+P T −−	+++++−	++P −−	+P T −−	++T −−	++P −−−	+P T −−	+P −−−	++P −−	++P −−
	1929	+P T −−	++P −−	+P P T −	+++P T	+++P T	P P T −−	+P P T −	++P T −	+P T −−	+++P −	++P T −	+++P T
	1930	+++P T −	+P T −−										
128	1926			++P −−	++P −−	++P −−	++P −−	++−−−	++−−−	++−−−	+−−−−	++P −−	++−−−
	1927	++−−−	+−−−−	++−−−	++P T −	++T −−	++T −−	+++T −	T T T −−	++T −−	P P −−−	++P −−	+P T −−
	1928	+P P −−	+++P T −	+P T −−	++P −−	++P −−	++P T −	++P T −	++P T −	++T −−	++P −−	++P T −	++P −−
	1929	+++P −	+++T −	+++P −−	++P T −	++P T −	++P T −	+++P −	+++P T	+P T −−	+++P T −	+P T −−	+++P T −
	1930	+++P T −	+P T −−										
205	1926			+P −−−	P −−−−	+++P −	+++−−	+P −−−	P P −−−	+++++−	+++P −	+++T −	++++P
	1927	+++−−	+++++−	+++P −	+++++	+++P T	+++P −	+++++	+++++	+++++	+++++	+++++	+++++
	1928	+++++	+++++	+++++	+++++	+++++	+P −−−	+++++	+++++	+++++	+++++	+++++	+++++
	1929	+++++	−−−−−	−−−−−	+++++	+++++	+++++	+++++	+++++	+++P −	+++++	+++++	Dead

TABLE XXV—*Group IID1. Originally positive and became suspicious*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
171	44	5	283 243 264 281 268	Aborted. *Br. abortus* isolated Dead at delivery	Slaughtered 10-2-29 Unprofitable	Negative
178	46	5	263 268 271 273 276		Living	Negative
191	42	3	198 251 280	Aborted Retained fetal membranes	Slaughtered 7-26-29	Positive
228	37	2	263 289		Sold. Pregnant	Negative

TABLE XXVI—*Agglutination reactions of group IID1*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
171	1926			++P P P	++++−	++++P	+++++	+++++	+++++	++++P	+++++	+++++	+++++
	1927	+++P P	+++−−	+++P−	+P−−−	+++T−	++P P T	++P P T	+++T	++P T−	+++P T	+++P T	++P P T
	1928	+++P T	+++++P	+++P−	++P T−	−−−−−	T−−−−	−−−−−	+P T−−	P T−−−	−−−−−	+P T−−	++P−−
	1929	+++P−	++P T−	++−−−	++P−−	+T−−−	+−−−−	+P T−−	++P T−	+P T−−	+P T−−	Dead	
178	1926			++P P−	+++−−	++++−	+++−−	++−−−	++−−−	+P−−−	+−−−−	P T−−−	++−−−
	1927	++P−−	+−−−−	P−−−−	+P−−−	+P T−−	−−−−−		+−−−−	+T−−−	T T−−−	P T−−−	P T−−−
	1928	−−−−−	P T−−−	++T−−	+P−−−	P P−−−	−−−−−	P T−−−	+P−−−	+T−−−	T−−−−	T−−−−	P T−−−
	1929	−−−−−	−−−−−	−−−−−	+T−−−	T−−−−		−−−−−	P T−−−		P T−−−	−−−−−	−−−−−
	1930	−−−−−	−−−−−										
191	1926			+++−−	+++P−	+P−−−	+++−−	+++P−	+++P−	+++++	+++++	+++++	+++++
	1927	+++++	++++−	+++++	++++++	P T T−−	++++T	+++P−	++++T	+++P T	+++P T	+++P T	+++++
	1928	+++++	+++P T	+P−−−	+P−−−	+P−−−	P T−−−	−−−−−	−−−−−	−−−−−	T−−−−	T−−−−	T−−−−
	1929	+P T−−	P T T−−	T P T−−	P T P−−	++++P T	−−−−−	−−−−−	Dead				
228	1926						++−−−	+++−−	+++−−	+++−−	+++−−	++−−−	T−−−−
	1927	P−−−−	P−−−−	−−−−−	+−−−−	T−−−−	−−−−−	T−−−−	T−−−−	−−−−−	T−−−−	P−−−−	T−−−−
	1928	−−−−−	−−−−−	+P−−−	P T−−−	T−−−−	−−−−−	+P−−−	P T−−−	+P T−−	+P T−−	+P−−−	+P T−−
	1929	P T−−−	T−−−−	T−−−−	−−−−−	T−−−−	−−−−−	T−−−−	P T−−−	−−−−−	−−−−−	−−−−−	Sold

TABLE XXVII—*Group IID2. Originally positive, became negative and then suspicious or positive*

ANIMAL	AGGLUTINATION TESTS	PARTURITIONS	GESTATION (DAYS)	TERMINATION OF GESTATION	REMARKS	HISTORY OF UDDER INFECTION
137	47	6	277			
			280			
			271		Living	Negative
			275			
			275			
			279	Now under treatment for sterility		

TABLE XXVIII—*Agglutination reactions of group IID2*

ANIMAL	YEAR	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
137	1926			+ + + + +	+ + + + +	+ P P − −	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ + + − −	+ + + + +	+ + + + +
	1927	+ + + + +	+ + + + +	+ + + + +	+ + + + +	+ P P T −	+ + + + +	+ + + + −	+ + + P T	+ + + + T	+ + + P T	P P T − −	+ + + P T
	1928	+ + + + +	+ + + + +	− − − − −	+ + + + P	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −	− − − − −
	1929	− − − − −	T	T P P P T	− P + P T	T P T − −		+ + + + +	− − − − −	− − − − −	− − − − −	− − − − −	+ + + P T
	1930	+ + P T −	− − − − −										

TABLE XXIX—*Summary of breeding records by groups*

GROUP	ANIMALS	ABORTED		PARTURITIONS	PARTURITIONS TERMINATING IN ABORTION	ABORTIONS (%)	COWS WITH RETAINED FETAL MEMBRANES		CASES OF RETAINED FETAL MEMBRANES		COWS BECAME STERILE		BR. ABORTUS ISOLATED FROM UDDER	
		No.	%				No.	%	No.	%	No.	%	No.	%
IA	15	3	20	42	7	16	4	26	5	12	1	6	0	
IB	11	9	82	57	20	35	5	45	7	12	6	54	7	63
IC	2	0		10			0				1	50	0	
IIA1	13	2	15	55	3	6	2	15	2	3	5	38	2	15
IIA2	13	5	38	42	6	14	3	23	3	7	1	8	1	8
IIA3	6	4	66	14	4	28	1	16	1	7	4	66	3	50
IIB1	15	9	60	35	10	28	2	13	2	5	10	66	11	73
IIB2	7	1	14	21	1	4	2	28	2	9	0		0	
IIB3	6	2	33	21	4	19	5	83	7	33	2	33	0	
IIC1	4	2	50	18	3	16	1	25	1	5	1	25	0	
IIC2	3	3	100	21	4	19	1	33	1	4	0		1	33
IID1	4	2	50	15	3	20	1	25	1	6	0		1	25
IID2	1			6			0				0		0	

TABLE XXX—*Summary of breeding records*

Animals grouped according to final agglutination reaction.* (See table II)

FINAL GROUPING	COWS	ABORTED		PARTURITIONS	PARTURITIONS TERMINATING IN ABORTION		BECAME STERILE	
		No.	%		No.	%	No.	%
Negative	39	11	28	123	17	13	3	7
Suspicious	26	6	23	107	10	9	8	28
Positive	35	25	71	127	38	30	20	57

*Negative includes groups IA, IIA2, IIB2, IIC1.
Suspicious includes groups IC, IIA1, IIB3, IID1, IID2.
Positive includes groups IB, IIA3, IIB1, IIC2.

NOTE

In considering the surprisingly high abortion rate in negative animals, the following facts should be borne in mind:

1. This group includes some old animals whose early agglutination history is unknown.
2. Some of the animals in this group were known to have heavy streptococcic infections of udder and fetal membranes.
3. In certain cases the cause of abortion was determined (*B. coli*, *Vibrio fetus*, Stretpococcus).
4. Some of the animals in this group gave suspicious, and some positive agglutination reactions at some time in their history of testing.

3. Three (23%) (cows 89, 232 and 236) were finally classed as positive.

B. Four were positive in 1:50 from five to ten months.

1. Three (75%) (cows 178, 179, 184) were finally classed as suspicious.

2. One (25%) (cow 19) was finally classed as positive.

C. One animal (cow 128) was positive in 1:50 more than ten months, and finally was classed as positive.

III. Fifty-four animals gave positive reactions in 1:100.

A. Thirteen were positive in 1:100 from one to five months.

1. Five (38%) (cows 130, 218, 225, 235 and 261) were finally classed as negative.

TABLE XXXI—*Summary of evidence of udder infection*

FINAL GROUPING	COWS	COWS FROM WHICH BR. ABORTUS WAS ISOLATED FROM UDDER	PERCENTAGE OF UDDER INFECTION
Negative	39	1*	2
Suspicious	26	3	11
Positive	35	22	62

*See cow 192, table XI.

TABLE XXXII—*Summary of incidence of retained fetal membranes*

FINAL GROUPING	ANIMALS	COWS WITH RETAINED FETAL MEMBRANES	PARTURITIONS	CASES RETAINED FETAL MEMBRANES	
				No.	%
Negative	39	10	123	11	8.9
Suspicious	26	9	107	10	9.3
Positive	35	9	127	11	8.7

2. Three (23%) (cows 167, 178 and 228) were finally classed as suspicious.

3. Five (38%) (cows 89, 128, 150, 211 and 215) were finally classed as positive.

B. Five were positive in 1:100 from five to ten months.

1. One (20%) (cow 214) was finally classed as negative. (See footnote, table XXXIII.)

2. Two (40%) (cows 184 and 187) were finally classed as suspicious.

3. Two (40%) (cows 190 and 208) were finally classed as positive.

C. Thirty-six were positive in 1:100 more than ten months.

TABLE XXXIII—*Summary of reactions and final grouping*

	Reactions	Animals	Final Grouping Negative		Final Grouping Suspicious		Final Grouping Positive	
			Number	Per Cent	Number	Per Cent	Number	Per Cent
Positive 1:50	1 to 5	13	7	54	3	23	3	23
	5 to 10	4			3	75	1	25
	10 or more	1					1	100
	Totals	18	7	38	6	33	5	28
Positive 1:100	1 to 5	13	5	38	3	23	5	38
	5 to 10	5	1*	20	2	40	2	40
	10 or more	36			7†	19	29	81
	Totals	54	6	11	12	22	36	67

*Cow 214 is the only animal on the records having more than three + reactions in 1:100, to become and remain negative. (See table XVIII)
†Cow 137 gave negative reactions for nine months, later becoming positive and then suspicious. (See table XXVIII)

1. Seven (19%) (cows 137, 159, 167, 171, 179, 191 and 209) were finally classed as suspicious. (See footnote, table XXXIII).

2. Twenty-nine (81%) (cows 19, 42, 44, 64, 95, 99, 147, 150, 151, 162, 166, 168, 174, 186, 189, 195, 198, 203, 204, 205, 211, 215, 216, 222, 223, 231, 233, 237 and 238) were finally classed as positive.

Conclusions

The foregoing data seem to warrant the following conclusions:

1. Positive animals had two and one-half times more abortions than negative animals.

2. Sterility was four times more frequent in suspicious animals, and eight times more frequent in positive animals, than in those with a negative history.

3. Retention of the fetal membranes occurred with almost equal frequency in negative, suspicious and positive groups.

4. A majority of positive animals (62 per cent) and an appreciable number of suspicious animals (11 per cent) had *Br. abortus*-infected udders.

5. Of animals positive in 1:50, 38 per cent became negative, 33 per cent became suspicious and 28 per cent became positive.

6. Of animals positive in 1:100, 11 per cent became negative, 22 per cent became suspicious and 67 per cent became positive.

7. Of animals positive in 1:50 or 1:100 for five to ten months, only 2 per cent became negative.

8. Animals positive in 1:50 or 1:100 more than ten months, did not become permanently negative.

Reference

[1]Huddleson, I. Forrest, and Abell, E.: Rapid macroscopic agglutination for the serum diagnosis of Bang's abortion disease. Jour. Inf. Dis., xlii (1928), p. 242.

Convention Notes

More than fifty applications for membership were filed during the meeting.

Three states west of the Mississippi River were not represented: Arizona, New Mexico and Nevada.

Every state east of the Mississippi River was represented at the meeting, with the exception of four New England states: Maine, New Hampshire, Vermont and Connecticut.

The conference of delegates from state and provincial associations, held Monday evening, was unusually well attended. Considerable interest was shown in the proposed plan for affiliating these organizations with the A. V. M. A.

THE VETERINARY HOSPITAL

With Special Reference to Accommodations for Small Animals

By HORST SCHRECK, *El Paso, Texas*

(Continued from page 179, August issue)

KENNEL DIMENSIONS

A few kennels will be needed 3′ x 6′ x 3′6″ (size 1).* These large kennels will also serve two congenial dogs of the same ownership. A kennel 3′ x 3′ x 3′ (size 2) will be found most generally satisfactory for the largest number of dogs, while 2′6″ x 2′8″ x 2′0″ (size 3) cages will be satisfactory for cats, puppies and the small dog breeds. The number of each size will depend largely on the needs of each individual hospital. It has been found that a kennel deeper than 3 feet is not practical from a cleaning standpoint and also due to the fact that the patient in such a kennel is enabled to dodge the grasping hand.

Aside from the question of the size of the kennels, we may classify them according to purpose or use. We have:

General all-accommodation kennels.
Cat cages.
Boarding-dog tie-kennels.
Skin-disease tie-stalls.
Pneumonia kennel.
Rabies kennel.
Drying kennel.
Destroying kennel.
Cages for rabbits and guinea pigs.
Movable kennels.

In smaller hospitals we would naturally not expect to find all these different types of kennels. In the first place, there would not be enough room for them and again, we would not have sufficient use for them to warrant their installation. However, every hospital should be equipped with at least one rabies kennel and preferably one or two skin-disease kennels.

In constructing a kennel we must strive to produce the impression on the owner, that his pet is as well taken care of as he would be at home under similar circumstances. The kennel must be large enough for the animal contained, not over-sized,

*The first dimension given is the depth, the second is the length, and the third is the height.

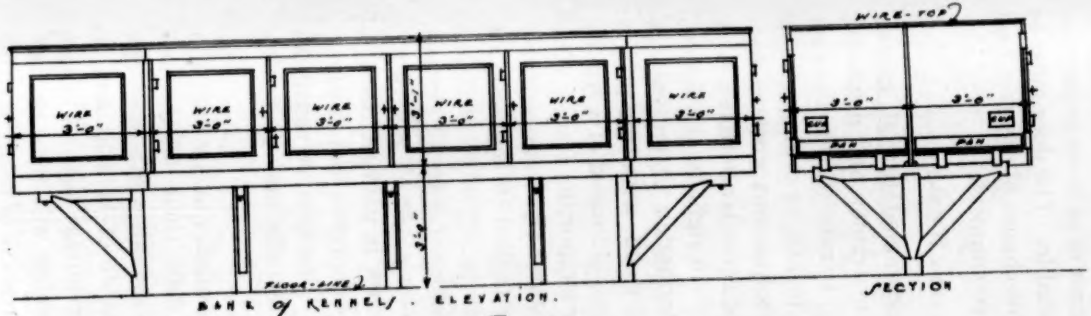

WIRE-TOP
WIRE 3'-0"
WIRE 3'-0"
WIRE 3'-0"
WIRE 3'-0"
WIRE 3'-0"
WIRE 3'-0"
3'-0"
FLOOR-LINE
3'-0"
3'-0"
3'-0"
DOOR
DOOR
RUN
RUN
BANK of KENNELS - ELEVATION.
SECTION

FIG. 7.

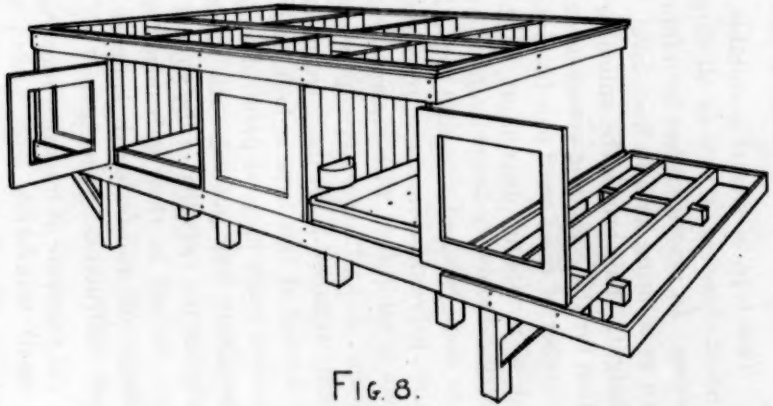


FIG. 8.

but just large enough and high enough, so that the animal's head can be raised to administer medication. The kennel must be so located as to be easily accessible to the kennelman for cleaning, and constructed in such a manner as to be as impervious to infection as possible and readily washable. The door must be securely locked to forestall every possible chance of escape. Last but not least, the kennel cost must be economical.

The General Accommodation Kennel

This type of kennel is suitable for accommodating all general hospital cases, as well as all surgical, maternity and distemper cases. In practice it has been found best to raise it from the floor so as to bring its own floor-level to 30 inches, or ordinary table height. This brings the animal within convenient working position for the attending veterinarian. The attendance that a large number of cases need can be given to them right in the kennel, at a minimum discomfort to the veterinarian or patient. A very satisfactory kennel can be built of wood, with iron grill in the door, exposed sides and top. The floor is formed of a removable, galvanized-iron pan, resting on the supporting frame. This pan is reinforced with channel iron, is water-tight and 4 inches deep. It and the water-cup, which is attached to the wall, are easily removable for daily cleansing, disinfection or sterilization. A kennel of this type is best built in banks of 4 to 12 kennels. Several coats of good paint of a light color cover all woodwork, previously well puttied. All the grill work is painted black to enhance the visibility of the animal. The floor-pan and drinking-cup are left in their galvanized state. Brackets of cantilever design, or wall-fastened, support the kennels and give little floor obstruction. (See figure 7.)

Cat cages are of the same construction, smaller in size and with a small transverse shelf added to satisfy their off-the-floor craving. (See figure 8.) The door opens the full width to permit removal of the pan. Since a large number of kennels are, or should be, of uniform size, we have the advantage that all kennel floor-pans will fit any kennel. The cleaning will thus be considerably simplified, allowing a moderate reserve of clean pans for emergencies. Another similar type of kennel is constructed with a stationary pan, with drainage attached to it, leading to a properly located waste-pipe. I doubt if this extra expensive plumbing installation is worth its cost.

The built-in kennel is constructed with terra cotta tile, brick,

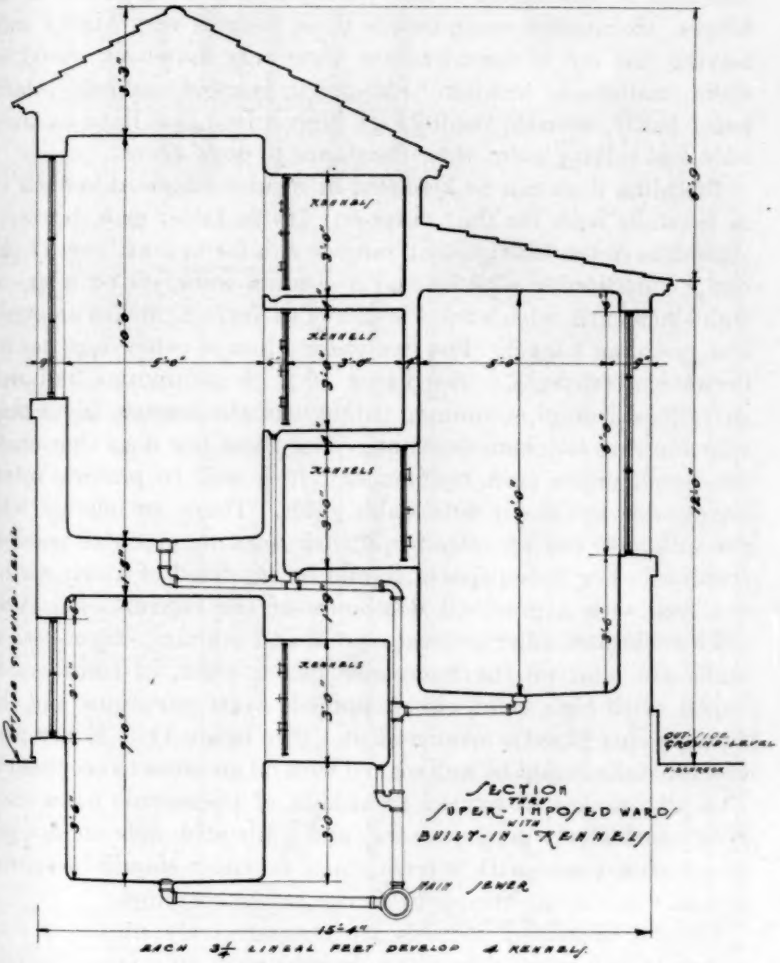

KENNELS
KENNELS
KENNELS
KENNELS
OUTSIDE GROUND-LEVEL
OUTSIDE GROUND-LEVEL
SECTION THRU SUPER-IMPOSED WALL WITH BUILT-IN KENNELS
MAIN SEWER
15'-4"
EACH 3½ LINEAL FEET DEVELOPS 4 KENNELS

FIG. 9.

gypsum block or wire lath and covered with hard plaster, vitrified tile or glazed brick. Drainage is maintained by a slight slope of the kennel floor to front or back, the waste material being taken care of by a small gutter leading to a waste-pipe. (See figure 9.) The door is constructed of heavy strap iron and hung from set-in hinges. Sanitation recommends these kennels very highly and, leaving cost out of consideration, their only drawback would be their unalterable location. All-metal kennels are noisy, hold paint badly, corrode readily, are high priced and have as their only real talking point their resistance to dogs' teeth.

Boarding dogs can be kenneled in regular standard kennels or in tie-stalls built for that purpose. In the latter case, boarders should have the liberty of an outside run for at least part of the day. Partitioning with framed iron mesh work, set on a six- or eight-inch curb, with a total height of seven feet, makes an excellent boarding kennel. The free observation of other dogs has its decided advantages. (See figure 10.) A continuous half-inch galvanized iron pipe, running through all the kennels, gives play to a slip-ring for chain-fastening. For those few dogs that chafe too much under such confinement, it is well to provide interchangeable and easily detachable gates. There are clients who are willing to pay an extra fee, if their dogs are given all possible freedom. For these, spaces should be provided of about six by ten feet with a practical dog-house of the conventional type. Such enclosures must be escape-proof and sanitary. Skin-disease stalls are built on the removable bench order, of considerable depth, with high, solid, close-matched, wood partitions and the running-ring pipe-tie arrangement. (See figure 11.) Every part of these stalls should be well soaked with oil previous to occupancy. The advantages of oxygen treatment of pneumonia cases have been conclusively demonstrated, and while such aids are not yet in common vogue with veterinarians, no effort should be spared to add this therapeutic agent to the armamentarium.

Oxygen inhalation kennels are comparatively simple in construction and most certainly a decided asset to a large hospital treating a large number of such cases. The circulation of oxygen in the air-tight chamber is maintained by continuous exhaust of all exhaled air by an electric suction-pump, thus allowing oxygen to stream in in regulated quantities, after having passed through ice-cooled chambers. Control over internal air-pressure is maintained by adjustment of the oxygen valve according to the reading on the air-pressure gauge. This kennel should best stand free

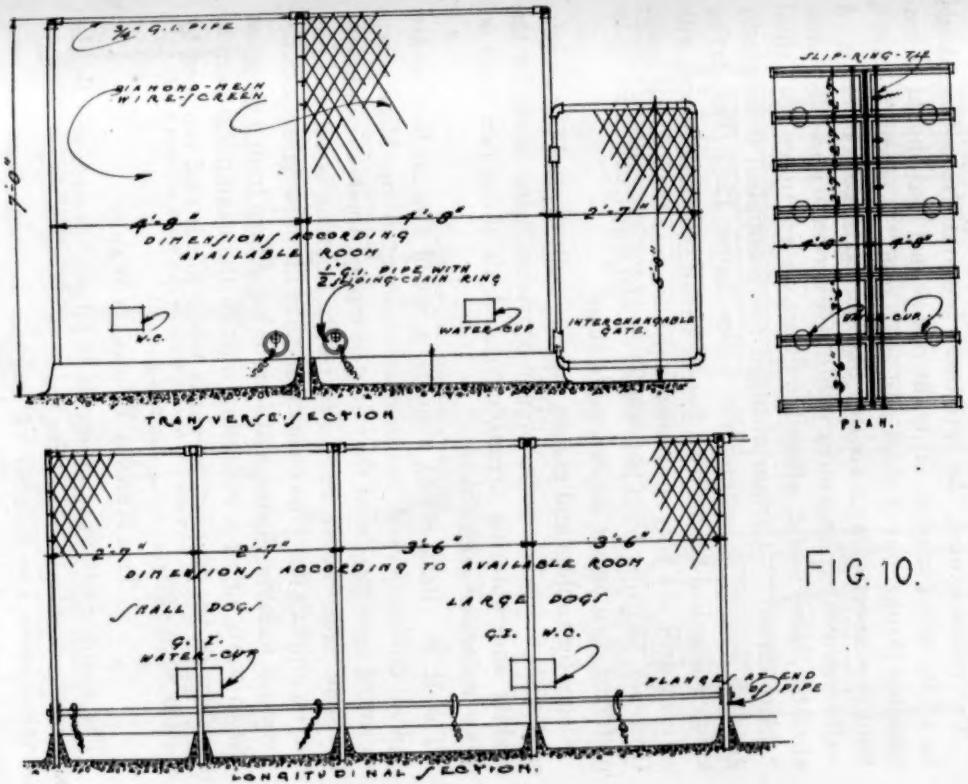

3/4" G.I. PIPE
DIAMOND-MESH WIRE SCREEN
7'-0"
4'-8"
4'-8"
2'-7"
DIMENSIONS ACCORDING AVAILABLE ROOM
1" G.I. PIPE WITH 2 SWING CHAIN RING
W.C.
WATER-CUP
INTERCHANGEABLE GATE.
TRANSVERSE SECTION
SLIP-RING-TYP.
4'-8"
4'-8"
PLAN.
2'-9"
2'-7"
3'-6"
3'-6"
DIMENSIONS ACCORDING TO AVAILABLE ROOM
SMALL DOGS
LARGE DOGS
G.I. WATER-CUP
G.I. W.C.
FLANGE AT END OF PIPE
LONGITUDINAL SECTION.

FIG. 10.

in the ward and have at least one unobstructed plate glass side. Noise should be avoided about such patients. Wood is an excellent material with which to construct these kennels. Provision must be made to give the pneumonia kennel an abundant fresh air supply after the oxygen treatment has been discontinued.

The rabies kennel, the veterinary hospital strong-box, should be all the word implies. It is the one kennel that should be constructed throughout of sheet and angle iron. It is well to have it built in connection with another like kennel, to allow cleaning. A well carried out arrangement would give access to an asphyxiation chamber, also useful where the owner desires to see the animal "chloroformed." Carbon monoxid gas is supplied from nearby gasoline engine or automobile. (See figure 12.) The drying kennel, a typical kennel with the floor removed and replaced with an open grill, is a very convenient adjunct near the bathing space. However, properly built benches of special construction are to be preferred if sufficient use warrants them.

Cages for rabbits and guinea pigs are built of steel frames covered with hardware cloth, proper provision being made for the feeding and watering arrangement. A dropping-pan rests on slides under the grilled floor.

The B. S. (Back Soon) kennels, a set of two to four or five typical double-decked kennels, are mounted on large, easy-operating casters, so that they may be moved about to convenient locations. (See figure 13.) Their only function is to take care of a few animals until the owner can make his personal appearance to consult with the doctor, until his or her return from a shopping tour to town, or as a collecting unit in the examining-room to be taken to the respective wards when filled, and replaced by another such unit.

The Kennel Rooms or Wards

The number of types of wards is largely determined by the size of the hospital. When we speak of size of hospital, we have in mind more the number of animals treated, than the area covered by the hospital. In large hospitals it will be a decided advantage to have several wards, taking care of different types of patients. In smaller hospitals, several wards, or all of them, will have to be consolidated into one ward or two. While we today very much question the possibility of confining distemper to a given ward, it is nevertheless good policy to make the attempt. Distemper will remain under better control, and the owners of

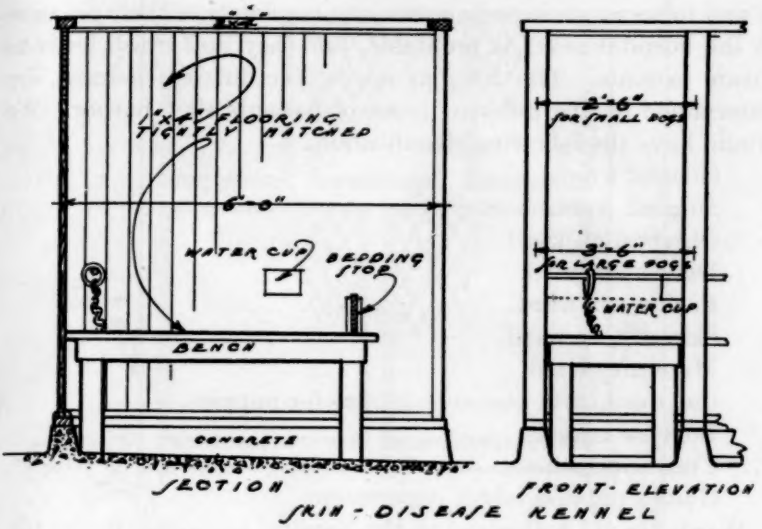

1"x4" FLOORING TIGHTLY MATCHED
6'-0"
WATER CUP
BEDDING STOP
BENCH
CONCRETE
SECTION
2'-6" FOR SMALL DOGS
3'-6" FOR LARGE DOGS
WATER CUP
FRONT-ELEVATION
SKIN-DISEASE KENNEL

FIG. 11.

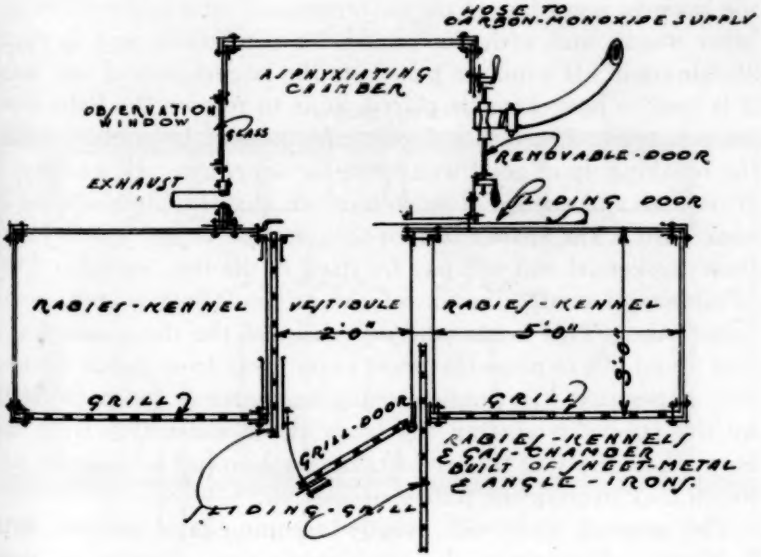

HOSE TO CARBON-MONOXIDE SUPPLY
ASPHYXIATING CHAMBER
OBSERVATION WINDOW
GLASS
EXHAUST
REMOVABLE-DOOR
SLIDING DOOR
RABIES-KENNEL
VESTIBULE
2'-0"
RABIES-KENNEL
5'-0"
3'-0
GRILL
GRILL
GRILL-DOOR
SLIDING-GRILL
RABIES-KENNELS & GAS-CHAMBER BUILT OF SHEET-METAL & ANGLE-IRONS.

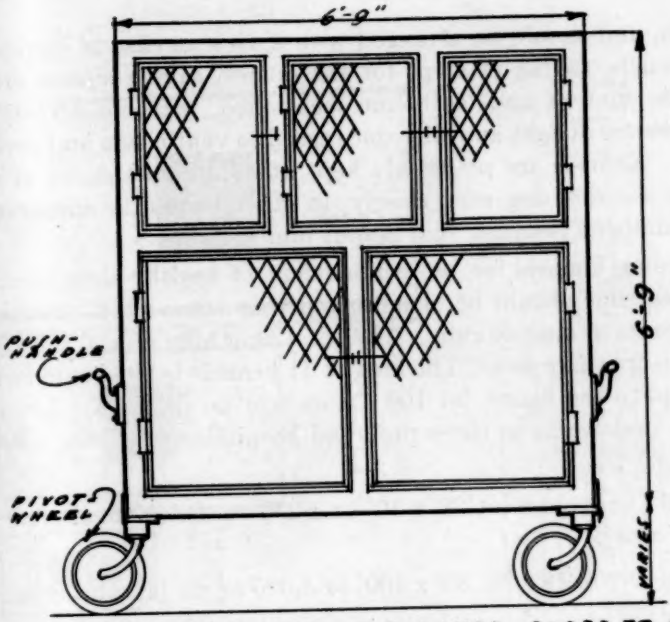
FIG. 12.

non-distemper dogs will be better satisfied. If at all possible, it is well to segregate boarding animals completely. Their presence in the hospital never is profitable, but they do furnish leads to future patients. The different wards, like different kennels, are determined by the different types of patients they harbor. We would have the following classification:

General ward.
Surgical ward.
Observation ward.
Distemper ward.
Pneumonia ward.
Skin-disease ward.
Maternity ward.
Cat ward, with accommodations for puppies.
Boarder kennels.
Transient kennels.
Guinea pig and rabbit department.

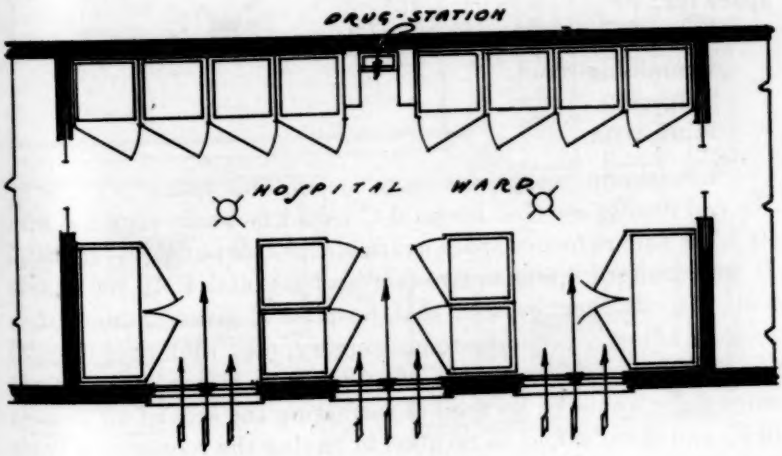
Wards should be located in the hospital according to the frequency of the work performed therein; their internal arrangement, according to the number of kennels; the arrangement of the kennels, according to the entrances and exits to corridors and other wards, and with due regard for ventilation and daylight illumination. If windows penetrate the largest part of any wall, it is best to have kennels placed so as to receive the light from the side. (See figure 14.) Every effort should be made to avoid the breaking up of good wall space by doorways. It is always a great time and energy saver to have an auxiliary drug-station in each ward. The space used for this purpose is just a little more than one kennel and will pay for itself in the time saved and the avoidance of contamination of the original drug source, many times over. This holds true especially of the distemper ward. One would like to place this ward as far away from public circulation as possible. By public circulation we mean the path taken by the public in visiting the hospital, as distinctive from the hospital circulation, the paths taken by hospital attendants and which may overlap the public circulation.

The general ward will usually accommodate patients with fractures. Alimentary diseases, respiratory diseases of non-infectious nature, circulatory diseases, imaginary diseases and, if no other facilities are provided, maternity cases and boarders. Foxes that are frequent patients at some hospitals are best taken care of in this ward. Such a ward may contain from 15 to 50



KENNELS ARE G.I. LINED. SLOPE TO BACK AND DRAIN COLLECTIVELY INTO PAIL.

FIG. 13.



ARROWS INDICATE DIRECTION OF LIGHT ENTERING WARD

FIG. 14.

kennels, and should be arranged with a view to ease of cleaning the kennels, saving of steps for the doctor, saving square area or cubic content and, as before mentioned, with consideration to the source of light and with due regard to ventilation and circulation. Kennels are preferably kept at minimum size, so as to confine the sick dog very closely, in other words, to encourage the recumbent position, that is, put him to bed.

Boarding kennels for the confinement of healthy dogs for an extended time should be of more generous size and, if possible, have access to outside runs. The total floor area of a 3′ x 3′ x 3′ kennel is 9 square feet. The area of 11 kennels is 99 square feet. To simplify the figure, let 100 square feet be the area. Let us assume that we have three proposed hospitals as follows: (See figure 15.)

Hospital C covers a lot 25′ x 50′ or 1,250 sq. ft. (Kennel area—8 per cent).

Hospital D covers a lot 50′ x 100′ or 5,000 sq. ft. (Kennel area—2 per cent)

Hospital E covers a lot 75′ x 150′ or 11,250 sq. ft. (Kennel area—0.88 per cent)

Let us assume that hospital D is large enough to give us the space for:

Reception-room.
Examining-room.
Surgery.
Work-room.

Kitchen and other necessary accountable space.

We can readily see that hospital C would be badly crowded with its 1,150 square feet of space available for this purpose. Hospital D would afford 4,600 square feet and hospital E 10,450 square feet. It can then be seen that hospital E gives us nine times roomier offices, examining-room, surgery, etc., than does hospital C. Or, some of the rooms in C would have to be consolidated, and since there would be no need of increasing the area of all kennels in E, and there would be no good in having the rooms even twice as large as D, there would remain the alternative of adding more kennels or building individual runs. Let us then assume that C has 11 kennels, D 75 kennels (becoming D1) and E 150 kennels (becoming E1). (See figure 16.) The alignment then would be as follows:

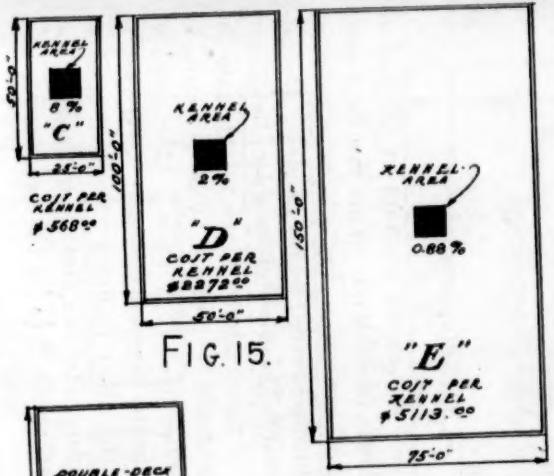


FIG. 15.

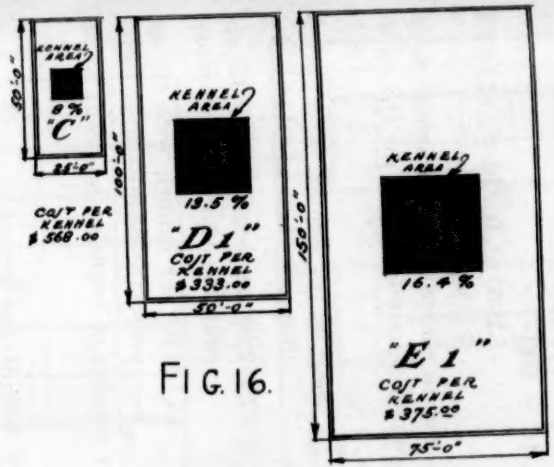


FIG. 16.

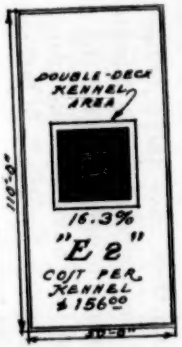


FIG. 17.

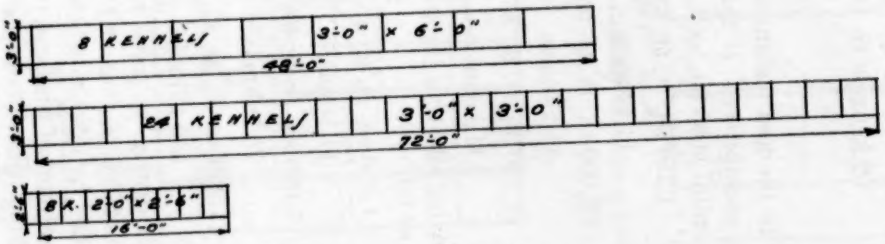


FIG. 18.

C—11 kennels @ 9 sq. ft. = 100 sq. ft. (8% of 1250 sq. ft.)
D1—75 kennels @ 9 sq. ft. = 675 sq. ft. (13.5% of 5000 sq. ft.)
E1—100 kennels @ 9 sq. ft. = 900
50 kennels @ 18 sq. ft. = 900
—— 1800 sq. ft. (16.4% of 11,250 sq. ft.)

In the first instance, the cost of construction per kennel would be prohibitive. If the assumed square-foot cost for the entire hospital is $5.00, we have:

C $\frac{\text{1,250 sq. ft. @ \$5.00}}{\text{11 kennels}}$ = \$568.00 per kennel, with its appropriate share of other rooms

D $\frac{\text{5,000 sq. ft. @ \$5.00}}{\text{11 kennels}}$ = \$2,272.00 per kennel, with its appropriate share of other rooms

E $\frac{\text{11,250 sq. ft. @ \$5.00}}{\text{11 kennels}}$ = \$5,113.00 per kennel, with its appropriate share of other rooms

After allotting the proper number of kennels, the result would be as follows:

C $\frac{\text{1,250 sq. ft. @ \$5.00}}{\text{11 kennels}}$ = \$568.00 per kennel

D1 $\frac{\text{5,000 sq. ft. @ \$5.00}}{\text{75 kennels}}$ = \$333.33 per kennel

E1 $\frac{\text{11,250 sq. ft. @ \$5.00}}{\text{150 kennels}}$ = \$375.00 per kennel

Evidently we have passed the happy medium at D1 and we should try to force E1 down to its level. This can be accomplished by double-decking all kennels, a step otherwise indicated on account of the large territory covered by the kennels. Double-decking is not always a good solution of this problem but is here assumed for the purpose of illustration. Since in this case the square area of the kennels would be reduced by 50 per cent, we would then have the equivalent of 75 kennels. Moreover, we have assumed that D1 has the correct sufficient total area to give us enough space for the other utilities. We find that we must then have the same space, that is 4,600 square feet, for this purpose, and to this add 900 square feet for the two-story kennel arrangement:

4,600 sq. ft.
900 sq. ft.
5,500 sq. ft. (or a lot 50′ x 110′).

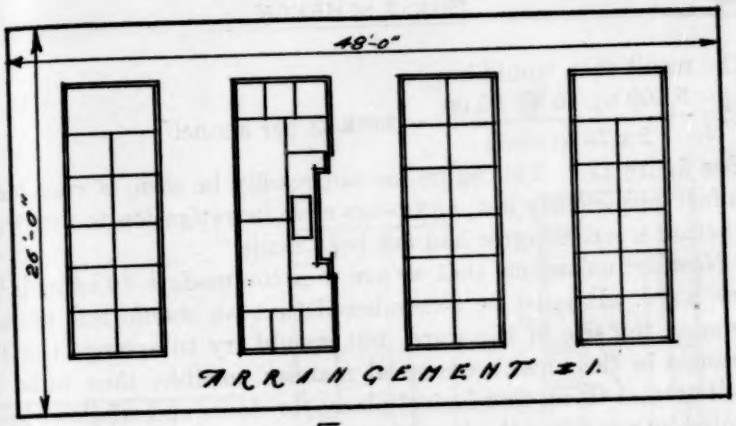


FIG 19.

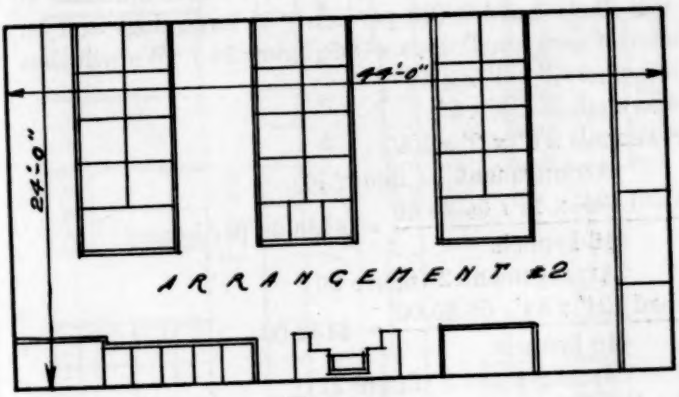


FIG. 20.

FIG. 21.

The result then would be:

$$E2 \quad \frac{5{,}500 \text{ sq. ft. @ } \$5.00}{2 \times 75 \text{ kennels}} = \$183.33 \text{ per kennel}$$

(See figure 17.) This figure, as can readily be seen, is very low, in fact suspiciously low, and bears close investigation to discover whether a serious error had not been made.

Now, let us assume that we are to accommodate 40 kennels in one ward. It must be remembered that we should not predetermine the size of the ward, but should try to arrange the 40 kennels in the most economical manner possible, then build a wall around them, find logical places for doors and windows and probably rearrange the kennels again, and from there on, let it be an element of the general plan. If we accommodate 40 dogs, we will likely have 8 (20 per cent) of large dogs and an equal number of very small dogs. (See figure 18.) We will then need:

8 kennels 3′ x 6′ x 3′6″
24 kennels 3′ x 3′ x 3′
8 kennels 2′6″ x 2′ x 2′8″

Arrangement 1 (figure 19)

$$\frac{\text{Ward } (26' \times 48') \text{ @ } \$5.00}{40 \text{ kennels}} = \$156.00 \text{ per kennel}$$

Arrangement 2 (figure 20)

$$\frac{\text{Ward } (24' \times 44') \text{ @ } \$5.00}{40 \text{ kennels}} = \$132.00$$

Arrangement 3 (figure 21)

$$\frac{\text{Ward } (24' \times 41'6'') \text{ @ } \$5.00}{40 \text{ kennels}} = \$124.80$$

Arrangement 4 (figure 22)

$$\frac{\text{Ward } (20' \times 46') \text{ @ } \$5.00}{40 \text{ kennels}} = \$115.00$$

Arrangement 5 (figure 23)

$$\frac{\text{Ward } (12' \times 72') \text{ @ } \$5.00}{40 \text{ kennels}} = \$109.25$$

In place of trying to rearrange the kennels further along the same line, let us resort to a drastic step and double-deck all kennels. This will effect a real economy of space. Since this method will reduce the number of square feet covered by kennels 50 per cent, we will then have (figure 24):

2 x 4 kennels 3′6″ x 3′ x 6′
2 x 12 kennels 3′ x 3′ x 3′
2 x 4 kennels 2′6″ x 2′8″ x 2′

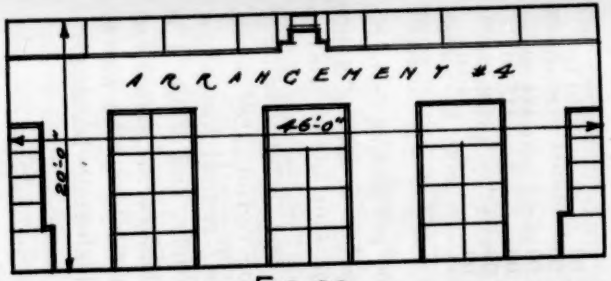


FIG. 22.

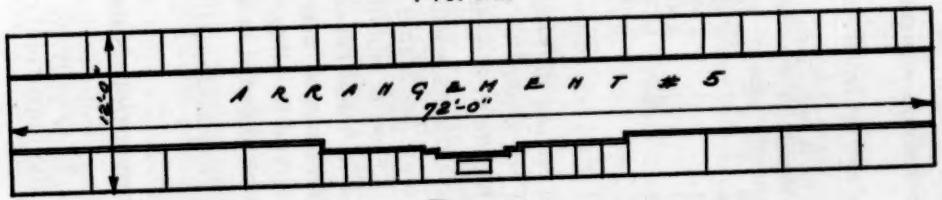


FIG. 23.

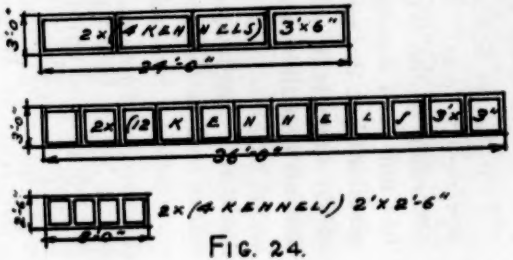


FIG. 24.

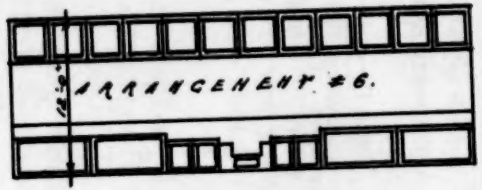


FIG. 25.

To avoid much cut-and-try method, just let us take the most economical arrangement of the examples gone before:

Arrangement 6 (figure 25)

$$\frac{\text{Ward (12' x 36') @ \$5.00}}{\text{2 x (20 kennels)}} = \$54.00$$

A summary of these findings is given in table II.

TABLE II—*Summary of costs per kennel with six different arrangements*

ARRANGEMENT	TOTAL AREA OF WARD (SQUARE FEET)	TOTAL COST OF WARD	COST PER KENNEL
1	1,246	$6,240.00	$156.00
2	1,056	5,280.00	132.00
3	998.4	4,992.00	124.80
4	920	4,600.00	115.00
5	874	4,370.00	109.25
6	432	2,160.00	54.00

These figures show a saving of $102.00 per kennel from the highest cost of $156.00 per kennel to the lowest cost of $54.00 per kennel; surely worthwhile looking into.

Five dollars has always been assumed as a nominal figure of square-foot cost. It may be higher or considerably lower, according to the type or class of building. Kennel cost proper has not been considered in these calculations, and while it is possible that the results presented may include the actual cost of the kennels in a low-grade building, it would not be wise to assume that they would be included in a high-grade building.

(*To be continued*)

Convention Notes

Dr. G. A. Roberts, of Christiansted, Virgin Islands, was in attendance and enjoyed the opportunity to shake hands with a host of old friends.

Dr. Kurt M. Wagener, of the Veterinary High School, Berlin, Germany, was an interested visitor at the meeting. Everyone enjoyed his paper, read at the Thursday evening general session.

Seven of the veterinary colleges were represented by their deans: Cary, of Alabama; Glover, of Colorado; Stange, of Iowa; Dykstra, of Kansas; Brumley, of Ohio; McGilvray, of Ontario; and Dick, of Pennsylvania.

A STUDY OF IMMUNITY OR RESISTANCE TO ASCARIDIA LINEATA (SCHNEIDER)*

By R. E. REBRASSIER *and* BRYCE R. MCCRORY

College of Veterinary Medicine, Ohio State University, Columbus, Ohio, in coöperation with the Ohio Agricultural Experiment Station, State Laboratories, Reynoldsburg, Ohio

INTRODUCTION

The greatest handicap to the poultry industry is disease and one of the major factors responsible for this condition is parasites. The nematode *Ascaridia lineata* (Schneider) is one parasite which is of common occurrence and causes a high mortality to young growing birds.

According to Herrick[1] a gradual resistance develops to *A. lineata* as chickens become older. Subsequently, Ackert and Herrick[2] reported that parasitized birds become resistant by the time they are three months of age. The reason for this resistance has not been definitely established. Zimmerman, Vincent and Ackert[3] found that resistance could be lowered by feeding a ration deficient in vitamin B. In 1927, Ackert, Fisher and Zimmerman[4] showed that when the ration of chickens lacked vitamin A, these nematodes were more numerous and much larger than in birds fed a normal ration.

Kolmer, Trist and Heist,[5] in summarizing their results of the complement-fixation test with sera of infested dogs, were of the opinion that the production of antibodies may occur after infestation of the intestines with common parasites. Schwartz[6] found that the host harboring parasites gave serological reactions which indicated that parasitic worms liberate products against which the host develops defense or immunity. He also mentions the probability that certain individuals may lack antilytic constituents in the blood and are then susceptible to the toxin, which other individuals are capable of neutralizing. Ramsdell[7] suggests the possibility of antibody response to parasitic infestation being of two types: (1) an antitoxin production, which diminishes with time following the removal of the parasites from the host and (2) a sensitization antibody which is apparent after the removal of the parasite.

*Received for publication, May 27, 1931.

Ransom, Harrison and Couch[8] stated that *Ascaris lumbricoides* and other closely related species, parasitic in various animals, have toxic properties. Ackert[9] reported that repeated bleeding of birds increases their susceptibility to *Ascaridia perspicillum* infestation. Herrick[1] observed that young chickens injected with blood sera from mature fowls and then infested by feeding embryonated *A. lineata* ova, harbored as many and as large parasites as did the control birds. However, the injected birds did not show the characteristic symptoms of parasitism as did the controls and they developed normally. This indicates the possibility of increasing the resistance of birds to *A. lineata* by artificial methods.

Analysis of the foregoing data indicates that antibody production is stimulated by intestinal parasites. An experiment was outlined to ascertain whether or not an immunity or resistance could be artificially produced, in the chicken, against *A. lineata* (Schneider).

METHODS

Day-old White Leghorn chicks were placed in a well-lighted, heated and ventilated poultry-house which was screened to prevent the admission of flies. These chicks were fed an all-mash growing ration.

At the beginning of the experiment the chickens were banded for identification, feed withheld for eighteen hours, after which they were individually weighed and separated in nine lots of approximately equal weight. The nine lots were kept under similar conditions, excepting as noted in the experiment. Records were kept of the feed consumed by each lot during the experiment, also symptoms and weight of each bird.

The *A. lineata* is a parasite inhabiting the small intestine of the chicken. It has been confused with several closely related species, and to be certain that we were dealing with *A. lineata* (Schneider), a definite classification was made with each collection of worms. The classification as outlined by Cram[10] was followed in this study.

The technic used in preparing and mounting the specimens was the alcohol-creosote method as modified by Reitz.[11] In this connection, it was found that if the alcohol, used for killing and fixation, was heated to 60-62°C., better preparations were obtained for mounting and study.

The parasite (*A. lineata*) has a direct development. With sufficient warmth and moisture the ova develop to the embryo

stage in about ten days, but with less favorable conditions, this period may be extended to weeks and months. Hatching normally occurs after the embryonated ova are ingested by a suitable host. According to Ackert,[12] larvae are found in the duodenum and jejunum within twenty-four hours following ingestion. They burrow into the mucosa and after a period of about twelve days, withdraw into the lumen of the intestine and mature in approximately six weeks.

The ova used for parasitizing were obtained by pressing the eggs from the uteri of adult *A. lineata* and culturing them in distilled water to which were added a few drops of 2 per cent formalin. The cultures were incubated for two weeks at which time the majority of the ova contained a coiled embryo. In parasitizing the birds, the embryonated ova were placed directly into the crop with the aid of a pipette.

The immunizing agent used in this experiment was an extract of the *A. lineata*. It was prepared according to the following technic: Immature and mature *A. lineata*, obtained from chickens were thoroughly washed in physiological salt solution and then placed in a solution consisting of sodium chlorid, 7.0 gms., phenol crystals, 4.0 gms. and distilled water, 1000 cc. When a sufficient number of parasites had been collected, they were again washed in physiological salt solution and ground in a mortar to a pulp-like consistency. The material was placed in a clay dish having a glazed outer surface and desiccated over sulphuric acid C. P. in the upper chamber of a vacuum jar. The jar was sealed with vaseline and its contents placed under partial vacuum for twenty-four hours, at which time the material was removed, finely powdered, and then replaced in the desiccator for an additional period of twenty-four hours. The powder was then stored in an air-tight glass container and kept at a temperature of 4 to 5° C. The extract was prepared for administering by suspending 0.05 gm. of the powder in 1 cc of sterile physiological salt solution.

Daily observations were made on the behavior of the chickens until the end of three weeks of parasitism. After killing the birds, the intestines were removed immediately, opened and their contents scraped into a receptacle containing warm tap water. The intestines and their contents were examined with a wide-field binocular microscope. The number and length of worms were determined and the condition of the intestines carefully observed.

EXPERIMENT

July 15, 1930, one hundred forty-four birds were selected and divided into three equal groups according to age. Group A contained birds 46 days of age; group B, birds 97 days of age; and group C, birds 131 days old. Each group was divided into three lots of 16 birds.

Birds in lot 1 of each group were injected, intraperitoneally, with 1 cc of *A. lineata* extract. July 19, 1930, each bird in lots 1 and 2, of each group, was given approximately 500 embryonated ova of *A. lineata*. Lot 3, of each group, was kept as a control, receiving neither embryonated ova nor extract. The embryonated ova were administered to the birds after they had undergone an eighteen-hour period of fasting, at which time each bird was weighed. This fasting period was considered essential in order to minimize the amount of food in the alimentary tract.

Throughout the experiment, all birds were fed the following ration:

Yellow corn (ground)	70 parts
Middlings	20 parts
Meat scraps	5 parts
Bone meal	2 parts
Cod-liver oil	1 part
Salt	1 part
Dried buttermilk	10 parts
Alfalfa meal	5 parts

A record was kept of the amount of feed consumed by each lot.

The experiment was terminated at the end of the third week of parasitism, as Ackert and Herrick[2] had shown that a rapid elimination of young worms begins at this time, and that the most serious effects were observed during the third week of parasitism.

At the close of the experiment, August 11, 1930, each bird was weighed, the feed having been withheld for a period of fifteen hours.

A noticeable difference in weight was observed in the birds in group A, which were 72 days old at the close of the experiment. In the birds of lot 1, which received both extract and ova, there was an average gain of 170.9 gms. which was a gain of 13.9 gms. per bird over those of lot 2, which received only ova, the average gain of which was 157 gms. In lot 3, the control birds, there was an average gain of 187.9 gms., this being a gain of 17 gms. more than in lot 1 and 30.9 gms. more than in lot 2.

The birds in group B, which were 123 days old, showed variations in weight similar to group A. Lot 1 made an average gain

of 214.7 gms., this being 40.9 gms. more than the average gain in lot 2, the average gain for this lot being 183.8 gms.

Lot 3, the controls, had an average gain of 217.4 gms., this being 2.7 gms. more than in lot 1 and 33.6 more than in lot 2.

Very little difference was observed in the average gain of the lots in group C. This probably was due to the fact that this group had reached an age whereby an immunity or resistance againse *A. lineata* had developed.[2]

A very noticeable difference in the amount of feed consumed per lot was observed in group A, while in group B it was less apparent and very little difference occurred in group C. In lots 1 and 2, group A, there was practically no difference in the average amount of feed consumed per bird. Lot 3 consumed an average of approximately 183 gms. more feed per bird than either of the other lots. Lot 1, group B, consumed 63 gms. more feed per bird than lot 2, and 107 gms. more than lot 3. In lot 1, group C, 95 gms. more feed per bird was consumed than in lot 2, and 62 gms. more per bird than in lot 3.

Autopsy revealed an enteritis in many of the birds. This was especially true of the birds of group A. In lot 1, birds 203, 207 and 212 showed a mild inflammation of the small intestine, while the remaining birds of this group did not show this condition.

In lot 2, birds 218, 220 and 223 revealed a mild enteritis and birds 221, 222, 225, 226, 229, 231 and 232 showed a very marked inflammation. All the birds in lot 3 were free of parasites and there was no evidence of enteritis.

In group B, lot 1, birds 7 and 15 had a mild enteritis, but the remaining birds in this lot did not show this condition.

In lot 2, group B, a mild enteritis was observed in the anterior portion of the small intestine in birds 17, 23, 27, 28 and 31. Bird 26 died July 29 and larvae of *A. lineata* were found embedded in the mucosa of the small intestine. The remaining birds of the lot and all in lot 3 revealed no evidence of enteritis.

In group C, lot 1, bird 57 showed a mild enteritis, and in lot 2, two birds (70 and 72) showed this condition. In lot 3, no evidence of inflammation was observed.

As shown in table I, group A, the average number of worms per bird was 2.2 in lot 1, and 0.25 in lot 2, and none in lot 3. The average length of worms in lot 1 was 1.88 mm. and in lot 2, 1.0 mm. In group B, as shown in table II, the average number of worms in lot 1 was 0.12 while in lot 2 it was 0.26, and none in

TABLE I—*Data from experiment testing the value of Ascaridia lineata extract as an immunizing factor in chicks forty-six days of age*

CHICK	WEIGHT (GRAMS)		NUMBER OF WORMS	AVERAGE LENGTH (MM.)	FEED CONSUMED
	JULY 19	AUGUST 11			
Group A, Lot 1 (extract and ova)					
201	198	Died 7-26	0	0	
202	139	286	0	0	
203	152	325	7	12.5	
204	150	273	0	0	
205	155	334	1	2	
206	172	332	0	0	
207	155	324	0	0	
208	144	329	0	0	
209	181	334	0	0	
210	148	314	2	6.5	
211	165	359	0	0	
212	144	304	3	6	
213	201	402	0	0	
214	159	340	0	0	
215	146	324	0	0	
216	155	330	31	1.22	10,320 grams
Aver.	156.4	327.3	2.2	1.88	674
Group A, Lot 2 (ova)					
217	155	187	0	0	
218	199	361	0	0	
219	168	340	1	11	
220	224	420	0	0	
221	187	361	0	0	
222	137	246	0	0	
223	174	320	0	0	
224	133	292	0	0	
225	205	405	0	0	
226	158	314	0	0	
227	185	328	3	5	
228	173	333	0	0	
229	179	375	0	0	
230	191	346	0	0	
231	177	348	0	0	
232	129	270	0	0	10,800 grams
Aver.	170.8	327.8	0.25	1	675
Group A, Lot 3 (control)					
233	170	354	0	0	
234	216	439	0	0	
235	199	446	0	0	
236	146	304	0	0	
237	168	344	0	0	
238	204	420	0	0	
239	206	434	0	0	
240	168	340	0	0	
241	144	307	0	0	
242	140	283	0	0	
243	155	320	0	0	
244	178	Died 8-7	0	0	
245	153	346	0	0	
246	187	403	0	0	
247	177	367	0	0	
248	170	319	0	0	12,000 grams
Aver.	173.8	361.7	0	0	758

TABLE II—*Data from experiment testing the value of Ascaridia lineata extract as an immunizing factor in chicks ninety-seven days of age*

CHICK	WEIGHT (GRAMS)		NUMBER OF WORMS	AVERAGE LENGTH (MM.)	FEED CONSUMED
	JULY 19	AUGUST 11			
Group B, Lot 1 (extract and ova)					
1	693	866	2	17.5	
2	763	1018	0	0	
3	889	1156	0	0	
4	702	956	0	0	
5	861	1010	0	0	
6	950	1289	0	0	
7	864	1199	0	0	
8	606	833	0	0	
9	530	772	0	0	
10	659	865	0	0	
11	607	800	0	0	
12	679	790	0	0	24,090 grams
13	584	829	0	0	
14	731	951	0	0	
15	512	698	0	0	
16	680	732	0	0	
Aver.	706.75	921.50	0.12	1.09	1505
Group B, Lot 2 (ova)					
17	833	1135	0	0	
18	921	1239	0	0	
19	1016	1283	0	0	
20	735	865	2	7.5	
21	860	994	0	0	
22	572	804	0	0	
23	543	707	2	11.5	
24	515	638	0	0	
25	529	775	0	0	
26	430	Died 7-29	0	0	
27	575	794	0	0	
28	759	937	0	0	
29	663	820	0	0	22,170 grams
30	781	809	0	0	
31	624	828	0	0	
32	538	707	0	0	
Aver.	695.13	889.0	0.26	1.26	1432
Group B, Lot 3 (control)					
33	668	954	0	0	
34	649	881	0	0	
35	842	1093	0	0	
36	686	1030	0	0	
37	842	1122	0	0	
38	817	1103	0	0	
39	515	736	0	0	
40	702	911	0	0	
41	692	890	0	0	
42	679	899	0	0	
43	738	976	0	0	
44	611	773	0	0	22,380 grams
45	697	813	0	0	
46	683	903	0	0	
47	655	781	0	0	
48	638	727	0	0	
Aver.	694.625	912.12	0	0	1398

TABLE III—*Data from experiment testing the value of Ascaridia lineata extract as an immunizing factor in chicks one hundred thirty-one days of age*

CHICK	WEIGHT (GRAMS)		NUMBER OF WORMS	AVERAGE LENGTH (MM.)	FEED CONSUMED
	JULY 19	AUGUST 11			
Group C, Lot 1 (extract and ova)					
49	985	1034	0	0	
50	1193	1353	0	0	
51	1325	1344	0	0	
52	1104	998	0	0	
53	1200	1485	0	0	
54	775	1073	0	0	
55	1104	1107	0	0	
56	1043	1115	0	0	
57	820	940	0	0	
58	774	910	0	0	
59	1021	1211	0	0	
60	782	759	0	0	
61	942	1210	3	3	
62	847	1002	0	0	
63	849	990	0	0	
64	1181	1451	0	0	23,130 grams
Aver.	996.56	1123.87	0.18	0.18	1445
Group C, Lot 2 (ova)					
65	809	913	0	0	
66	1280	1395	0	0	
67	758	887	0	0	
68	749	898	0	0	
69	904	1299	0	0	
70	1199	1180	1	7	
71	1132	1230	0	0	
72	1140	1356	1	10	
73	996	1205	0	0	
74	945	1183	1	5	
75	852	930	0	0	
76	938	1013	0	0	
77	985	1199	0	0	
78	798	786	0	0	
79	943	1053	0	0	
80	899	910	0	0	21,600 grams
Aver.	957.93	1089.81	0.18	1.37	1350
Group C, Lot 3 (control)					
81	1112	1169	0	0	
82	768	839	0	0	
83	1044	1009	0	0	
84	1243	1380	0	0	
85	1138	1222	0	0	
86	1077	1302	0	0	
87	1258	1451	0	0	
88	1296	1461	0	0	
89	725	859	0	0	
90	931	976	0	0	
91	820	912	0	0	
92	882	1075	0	0	
93	933	1065	0	0	
94	916	1096	0	0	
95	862	1063	0	0	
96	855	988	0	0	22,140 grams
Aver.	991.25	1116.68	0	0	1383

lot 3. The average length of the worms in lot 1 was 1.09 mm. and in lot 2, 1.26 mm. As shown in table III, group C, the average number of worms was 0.18 in both lots 1 and 2, while no worms were found in lot 3. The average length of the worms in lot 1 was 0.18 mm., while in lot 2 it was 1.37 mm.

The foregoing data show that in the chickens of group A there was a difference in the three lots as to the average gain in weight. Lot 3 showed a much greater average gain than did either of the other lots. The birds in lot 1, which received extract and ova, showed a decided gain over the birds in lot 2, which received only ova. Somewhat similar results were obtained with the birds of group B, while there was no noticeable difference in the average gain in group C. Considering the average gain in weight shown in the lots which contained the younger birds, it appears that the administration of the extract had some value in protecting them against the effects of parasitism.

A greater variation occurred in the average amount of feed consumed per lot in group A than in groups B and C. The fact that lots 1 and 2 of group A consumed less feed than lot 3 might be attributed to the effects of parasitism, since the birds of group A were of an age at which *A. lineata* causes the most damage.

Likewise the data relative to enteritis indicate that the administration of the worm extract may be a factor in protecting against the effects of *A. lineata*. Enteritis was not observed in any of the birds of the control lots, while this condition did exist in those which received ova. Enteritis was not so prevalent nor so marked in the birds which received extract as in those which received only ova. This is especially evident in the birds of group A. More birds showed enteritis and its severity was greater in lot 2 than in lot 1.

Summary

1. An experiment with 144 chickens was conducted to ascertain if the administration of an extract of the nematode *Ascaridia lineata* (Schneider) would increase their resistance to this parasite.

2. The greatest variation in the average gain per bird was found in group A. The controls made the most gain. The lot receiving the extract and ova made a greater gain than the lot which received only ova. In group B, similar gains were made but the difference was not so marked.

3. A greater variation in the average amount of feed consumed was observed in group A. The parasitized birds of groups A and B did not consume so much feed as did the controls.

4. An enteritis was present in a majority of the birds in lot 2, group A, which received only embryonated ova, while in lot 1, which received extract and ova, only a small number showed this condition and it was of a milder type. In lots 1 and 2, group B, similar results were observed, but there were not so many affected as in group A. In group C, this factor was not significant. No birds in control lots showed this condition.

5. The birds in lot 1, group A (extract and ova), contained a greater number of worms than was found in lot 2 which received only ova, and the average length was about the same in each lot. There was no significant difference in this respect between these two lots in groups B and C.

REFERENCES

[1]Herrick, C. A.: Studies on the resistance of the chicken to nematode *Ascaridia perspicillum*. Amer. Jour. Hyg., vi (1925), pp. 153-172.
[2]Ackert, J. E., and Herrick, C. A.: Effects of the nematode, *Ascaridia lineata* (Schneider), on growing chickens. Jour. Parasitol., xv (1928), pp. 1-13.
[3]Zimmerman, N. B., Vincent, L. B., and Ackert, J. E.: Vitamin B, a factor in the resistance of chickens to *Ascaridia perspicillum* (Rud.). Abst. in Jour. Parasitol., xii (1926), 3, p. 164.
[4]Ackert, J. E., Fisher, M. L. and Zimmerman, N. B.: Resistance to parasitism affected by the fat-soluble vitamin A. Abst. in Jour. Parasitol., xiii (1927), 3, p. 219.
[5]Kolmer, J. A., Trist, M. E., and Heist, G. D.: Complement fixation in intestinal parasitism. Jour. Inf. Dis., xviii (1916), pp. 88-105.
[6]Schwartz, B.: Hematoxins from parasitic worms. Jour. Agr. Res., xxii (1921), pp. 379-432.
[7]Ramsdell, S. G.: A note on the skin reaction in Taenia infestation. Jour. Parasitol., xiv (1927), p. 102.
[8]Ramson, B. H., Harrison, W. T., and Couch, J. F.: Ascaris sensitization. Jour. Agr. Res., xxviii (1924), p. 577.
[9]Ackert, J. E.: Resistance of chickens to Ascaridia. Jour. Parasitol., xii (1925), p. 163.
[10]Cram, E. B.: Bird parasites of the nematode sub-orders Strongylata, Ascaridata, and Spirurata. Smithsonian Institution, U. S. National Museum, Bul. 140 (1927).
[11]Rietz, J. H.: Technic for permanent balsam mounts of small nematodes. Jour. A. V. M. A., lxxi (1927), n. s. 24 (4), pp. 473-473.
[12]Ackert, J. E.: On the habitat of *Ascaridia perspicillum* (Rud.). Jour. Parasitol., x (1923), p. 101.

Broadcasting Program

The Committee on Broadcasting of the New York State Veterinary Medical Society has announced the following schedule for September:

September 2—"The Importance of Healthy Animals in Milk Producing Herds," Dr. H. G. Hodges, Borden's Farm Products Company, New York.

September 9—"Variations in Butter Fat Content of Milk and Influencing Factors," Dr. C. R. Roberts, Sheffield Farms Company, New York.

September 16—"Diseases of Sheep," Dr. L. H. Tripp, Albany County Veterinarian, Albany.

September 23—"Poisoning in Farm Animals," Dr. C. A. Fehr, former president of the Hudson Valley Veterinary Medical Society, Hopewell Junction.

September 30—"Bringing in the Cows," Dr. George A. Knapp, former president of the New York State Veterinary Medical Society, Millbrook.

These broadcasts are now given on Wednesday of each week from 12:20 to 12:30 p. m., E. S. T., from Station WGY, Schenectady, N. Y.

INFECTION OF PIGS THROUGH THE SKIN WITH THE LARVAE OF THE SWINE KIDNEY WORM, STEPHANURUS DENTATUS*

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INTRODUCTION

While a number of workers have recorded observations on the morphology of the kidney worm, *Stephanurus dentatus*, and on its distribution in its swine host, and have noted various lesions which this parasite is capable of producing in the organs and tissues which it invades and with which it comes in contact, comparatively little was known concerning the life history of this parasite until the publication of two papers by Bernard and Bauche[1,2] on the portals of entry of the worm into the body of its host. Previously Tayler[3] had expressed the opinion that Stephanurus larvae gained entrance into the body of hogs "either through contaminated drinking water or food." Hellemans[4] considered the oral route to be the normal path of entry of Stephanurus larvae into the body of their host. While this latter investigator did not overlook the possibility of percutaneous infections, and even speculated on the course of migration of the larvae from the skin to various organs, including the kidneys and perirenal fat, he did not carry out any experiments to demonstrate the possibility of percutaneous infection.

According to Bernard and Bauche,[2] Pecaud[5] expressed the opinion that pigs became infected with Stephanurus as a result of swallowing the infective larvae of the parasites with food. It remained for Bernard and Bauche to announce that they had succeeded in experimentally demonstrating infections of pigs with *Stephanurus dentatus* as a result of skin penetration by the infective larvae. According to these investigators, Stephanurus larvae penetrate the skin of pigs readily, and this path of entry into the host is apparently the normal one, for it results in the development of perirenal and periureteral cysts, which contain

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mature worms. Administration of larvae by mouth, according to these writers, does not result in infections of the urinary organs but results in liver infection with cirrhotic hypertrophy of the liver in which organ, however, the parasites fail to reach fertile maturity.

In the course of investigations on the life history of *Stephanurus dentatus*, which have already been published in abstract,[6,7,8,9] the writers carried out rather extensive experiments with a view to checking the work of Bernard and Bauche, and determining (1) whether the infective larvae of this parasite are actually capable of penetrating the skin of swine, (2) whether the liver and organs other than the kidneys, perirenal fat and adjacent tissues and organs, actually escape infection with kidney worms when the larvae gain entrance into the body of swine through the skin, and (3) whether the kidneys, perirenal fat and adjacent tissues actually escape infection when pigs are infected by the oral route. The writers' findings with reference to percutaneous infections, which are at variance with those of Bernard and Bauche, are given in this paper. A paper dealing with Stephanurus infections produced as a result of oral administration of larvae will be published later. As has already been stated, however, by the writers,[7,8] when the larvae were given by mouth they worked their way to the liver, and occasionally to the lungs, as well as to other tissues and organs. Following infections with Stephanurus larvae *per os*, the worms reached the renal region rather late in the course of the life cycle of the parasites, as they did when larvae were administered percutaneously.

Methods Used in Experiments

Mature kidney worms collected from the perirenal fat and adjacent organs and tissues were chopped up with a pair of fine scissors to liberate the eggs. The chopped-up material was thoroughly mixed with moist animal charcoal to which a small quantity of boiled fecal filtrate or boiled fecal material obtained from pigs, sheep or other domestic animals, was sometimes added. The larvae usually developed normally either in charcoal containing no fecal material, or in charcoal containing fecal material.

The charcoal mixture containing the worm eggs was placed in small glass jars, Petri dishes, or other suitable glass containers, which were rather loosely covered in order to permit

ready access of air. The cultures were kept at room temperature for a week or longer before the larvae were isolated from them for infection experiments. When the larvae which emerged from the eggs reached the infective stage, they climbed up the sides of the glass container in which the culture material was kept, and usually moved about very actively in the films of moisture which settled on the walls of the glass. This upward migration indicated that the larvae had attained the infective stage.

In order to carry out the infection experiments described in this paper, the larvae were isolated from the cultures with the aid of the Baermann apparatus, and then concentrated in water. A small quantity of water containing large numbers of larvae was used in the various experiments on infection through the skin described in this paper.

The pigs used in the following experiments were from a stock of experiment pigs which has been free from kidney worms throughout its history; the sows were kept in an uninfected area, and the pigs were kept in clean pens. The pigs previously killed served as checks on those used in the experiments which follow.

Details of Experiments

Bernard and Bauche[2] noted that following the repeated exposure of the unshaved skin of swine, from which the hair had been clipped, to infective larvae of *Stephanurus dentatus*, raised papules appeared on the skin, resembling those produced by the larvae of human hookworms on the skin of susceptible subjects. Bernard and Bauche state that the papules did not become pustular and disappeared after a time. The writers' experiments, designed to determine whether the infective larvae of *Stephanurus dentatus* can penetrate the unbroken skin of swine, are given below.

1. Effects of Placing Larvae on the Intact Skin

Experiment 1: Two pigs from the same litter, about six weeks old, were exposed to a percutaneous infection with the larvae of *Stephanurus dentatus* as follows:

On February 24, 1927, a rich culture containing several thousand infective larvae was placed on the most tender portions of the abdominal skin of two pigs and the animals were held under restraint until the water containing the larvae had evaporated. No skin reaction was noted. On the following day, this procedure was repeated with another rich culture of infective larvae; no skin reaction was noted in either of the two pigs.

Pig 1 was killed April 19, 1927, fifty-seven days after it had been exposed to a percutaneous infection, and pig 2 was killed May 7, 1927, seventy-three days after it had been exposed to a percutaneous infection. Postmortem examination of these pigs failed to reveal lesions or worms in the lungs, liver, kidneys, and other organs which are commonly invaded by these parasites. Larvae obtained from the same cultures that were used in this experiment were fed to two pigs from the same litter as those used in the above tests, and on portmortem examination 49 and 71 days, respectively, after oral administration of the larvae, marked lesions and heavy worm infestation were found in various thoracic and abdominal organs of these animals.

Experiment II: Another experiment designed to determine whether the infective larvae of *Stephanurus dentatus* can penetrate the intact skin of swine, was carried out as follows:

Two pigs from the same litter, 27 days old, were exposed to an infection by placing infective larvae on the skin on June 18, 1927, under conditions similar to those described in experiment I. These animals remained with the sows until they were weaned at the age of about eight weeks, and subsequent to weaning they were kept in a small field pen having a concrete floor. On August 31, 1927, seventy-four days after exposure to this experimental infection, the two pigs were killed and necropsied with the following results:

Pig 3: Conditions of carcass fair. Surface of liver smooth and shining; several light areas present on upper surface of liver and two small circumscribed areas, about the size of a pea, on lower surface of liver. On section, the latter areas appeared to be cirrhotic, the connective tissue proliferation being most abundant immediately under the capsule. Other abdominal and thoracic organs appeared normal. No kidney worms were found on macroscopic inspection or in press preparations, in any of the thoracic and abdominal organs, including the portal vein, which were examined.

It should be noted that pig 3 had a congenital cleft palate, and had difficulty in eating; this probably accounts for its general condition which was inferior to that of pig 4.

Pig 4: General condition of carcass very good. Surface of liver smooth and shining; liver light in color and with many light, firm areas, about one centimeter in diameter, evenly distributed over the surface. On section these areas showed what appeared

to be an increase in the interlobular connective tissue; this increase was more abundant immediately under the capsule than in the deeper portions of the hepatic tissue, where it diminished gradually. Other organs apparently normal. No worms found on macroscopic examination or in press preparations, in any abdominal and thoracic organs and tissues, including the portal vein.

Two pigs from the same litter as pigs 3 and 4 were given Stephanurus larvae by mouth at the same time that the latter were exposed to a percutaneous infection; the larvae were obtained from the same cultures as those used in connection with the skin penetration tests described above. The oral administration of larvae resulted in marked lesions and worm infestations found at necropsy in various abdominal and thoracic organs on August 30, 1927.

A third experiment was conducted as follows:

Experiment III: On July 6, 1929, about 400 infective Stephanurus larvae were placed on the intact abdominal skin of two pigs weighing 26 and 28½ pounds respectively. These pigs (5 and 6) were then about a month old. These animals remained with the sow until weaning time, approximately four weeks after experimental exposure, following which they were transferred to a field pen having a concrete floor, and remained there until April 4, 1930, a period of nine months, less two days, at the end of which the pigs were killed and necropsied. The lungs, liver, spleen, kidneys and other abdominal and thoracic organs appeared normal and no kidney worms were found. Several pigs, from the same litter as pigs 5 and 6, were given infective kidney worm larvae by mouth; the latter were killed about a week earlier than pigs 5 and 6 and were found to harbor numerous kidney worms with associated lesions in various thoracic and abdominal organs.

It is evident from the above experiments that the intact skin of young pigs, which is considerably more tender than that of older pigs, and presumably less resistant to attacks by nematode larvae capable of penetrating the skin, was not penetrated by infective larvae of *Stephanurus dentatus*. As already noted, larvae obtained from the same cultures used in these experiments produced typical lesions and worm infestations in various organs and tissues of pigs to which they were administered by mouth, thus showing conclusively that the failure of Stephanurus larvae to penetrate the intact skin of pigs used in the above experiments

was not due to a possible lowered vitality of the larvae but rather to their inherent inability to penetrate the intact skin of pigs.

The liver lesions noted in pigs 3 and 4, though superficially resembling those produced by infestation with *Stephanurus dentatus*, are pathological changes of rather common occurrence in the livers of swine in various parts of the United States, including many northern states, such as Wisconsin, New York, and other states where kidney worm infestation in hogs is either infrequent or not known to occur. The lesions in question differ from those produced by Stephanurus in that the former are superficial, do not contain pus or caseous material and do not show direct evidence of worm infestation. The etiology of these liver lesions has not been determined, though larval ascarids migrating through the liver and possibly other swine parasites migrating through this organ, have been suspected by the writers of being the incitants.

2. Penetration of Stephanurus Larvae Through the Scarified Skin

In view of the failure of infective Stephanurus larvae to penetrate the intact skin of pigs as determined in the above tests, a series of experiments was conducted to test the ability of the larvae to penetrate the broken skin of pigs. The details of these experiments are given below.

Experiment IV: On July 2, 1928, a rich culture of infective Stephanurus larvae was placed on the scarified skin of each of six pigs. Scarification was produced with the aid of an arrowhead lancet, but no blood was drawn. The larvae placed on the scarified skin were obtained from cultures which were started on June 15, and isolated on July 14.

The first pig (6) of this lot was killed on September 12, 1928, or 72 days after artificial infection. This pig showed the following lesions and worm infestation at necropsy:

Carcass emaciated. Liver with numerous necrotic areas, and with interlobular connective tissue greatly increased; portal vein thrombotic, the thrombi being firmly attached to the wall of the vein and showing evidence of organization; small worms, some perforating the wall of the vein, found in association with thrombi; lumen of gastro-hepatic artery almost occluded by a partially organized thrombus; perivascular connective tissue greatly increased. Kidneys showed organized infarcts, but no worms found in association with these lesions; kidneys and adjoin-

ing tissues uninfested with kidney worms. Posterior vena cava thrombotic; immature worms found in association with thrombi in this blood-vessel; lungs normal.

A second pig (8) of this lot, killed October 9, or 99 days after experimental infection, showed the following lesions and worm infestation at necropsy:

Liver with nodules and linear lesions underneath capsule; these lesions mark the paths of kidney worms which have traveled in a horizontal direction underneath the capsule; half-grown kidney worms present in linear lesions and elsewhere under capsule and some of the worms partially extruded through capsule; surface of liver and adjacent area of diaphragm greatly roughened. Kidney worms present in pancreatic tissue, close to kidneys. No worms in kidneys and kidney fat. Many degenerated and a few live worms and associated lesions present in lungs; the worms found in the lungs about half the size of those found in liver. All worms recovered from this pig were found to be immature.

One pig (9) of this lot died on October 24 and another (10) died on October 25, or 112 and 113 days, respectively, after artificial infection. The results of the postmortem examination of these pigs are given below.

Pig 9: Marked peritonitis present in portal region and on surface of stomach. Diaphragm and omentum adherent to liver; worms abundant in a pus pocket formed in a portion of the diaphragm adherent to liver, which was swollen and contained numerous caseous lesions; worms present on surface of liver underneath the capsule; portal vein showed numerous scars and thrombi. Spleen enlarged and covered with fibrin; several worms present in the fibrinous exudate. Worms and caseous lesions present in perirenal fat; kidneys apparently normal; ureters congested. One worm found penetrating wall of duodenum, the anterior half of worm being embedded in the wall; wall of duodenum not punctured through to lumen. Worms and associated lesions present in psoas muscles; caseous lesions present along vertebrae in lumbar region. A marked adhesive fibrinous pleurisy present, the exudate containing a number of kidney worms; lungs showed caseation and fibrosis; worms in lungs of same size as those in liver. None of the worms recovered from this pig contained uterine eggs.

Pig 10: Peritoneal cavity full of fluid; peritoneum roughened by a deposit of fibrin; portion of peritoneum closely adherent to

right lobe of liver; part of diaphragm also adherent to liver. Worms were found in the following locations: diaphragm, connective tissue near rectum, in lumbar region, in lumbar muscles, in muscles along spinal column, along the course of the dorsal aorta in the region where this blood-vessel perforates the diaphragm; in perirenal fat and in fat adjacent to ureter; one worm found in the wall of the ureter but not perforating it; 3 worms found under the capsule of the kidney, and two found in a pocket which communicated with the pelvis of the kidney; 3 worms found in the wall of the stomach but not perforating the wall of this organ; many worms found in the mesentery along wall of duodenum; numerous worms found in the liver, many partially extruding through the capsule; worms present in periportal connective tissue; numerous worms and associated lesions found in lungs; two worms found penetrating wall of posterior vena cava, the anterior half of the worms extending through the wall; one large, partly organized thrombus present in vena cava. All the worms recovered from this pig were sexually immature.

Another pig (11) from this lot was killed April 2, 1929, nine months after experimental infection. The following lesions and worm infestations were noted:

Liver with numerous healed lesions but no worms recovered from lesions; gastro-hepatic artery thrombosed, its lumen almost completely obliterated; bands of connective tissue extend across lumen of portal vein; numerous half-grown worms embedded in periportal connective tissue. Worms present in perirenal fat, apparently not fully grown; lesions in kidney fat showed no fistulous tract to ureters; no worms in ureters; one half-grown worm present in pelvis of kidney. Many worms, not quite fully grown, in psoas muscle in kidney region; other abdominal organs apparently normal. Lungs showed a few healed lesions, most of them without worms; worms about one-fourth the size of adult worms recovered from lung lesions. Several half-grown worms found along course of aorta and esophagus in mediastinal space, these worms enclosed in pus pockets; wall of posterior vena cava roughened but not thrombosed; no worms in this blood-vessel. No egg-producing worms were recovered from this pig.

The last pig of this lot (12) died October 29, 1929, or 15 months and 27 days following experimental infection. The post-mortem findings were as follows:

Carcass greatly emaciated. Considerable fibrin deposit present on surfaces of stomach and diaphragm adjacent to liver; worms

present on surface of liver and in liver lesions; portal vein thrombotic; periportal connective tissue greatly increased and containing numerous worms. Worms present in perirenal fat and underneath kidney capsule; no worms in pelvis of kidney or in ureters. Parietal and visceral pleura hyperemic and covered with fibrin; worms found on surface of lungs and in cystic lesions of lung tissue; posterior vena cava thrombotic.

It is evident from the data presented in this section of the paper that the infective larvae of Stephanurus penetrated the scarified skin of pigs and then made their way to various organs and tissues of the body which are known to be susceptible to infestation with these parasites. It is important to note in connection with these experiments that pigs 7 and 8, necropsied 72 and 99 days, respectively, after exposure of the scarified skin to infective larvae, showed typical lesions in the liver and hepatic blood-vessels with worm infestations in the lesions; they also showed lesions in various abdominal and thoracic viscera, except in the kidneys and adjacent tissues. The organized infarcts found in pig 7 were not associated with Stephanurus infestation. Two pigs (9 and 10) which were necropsied 112 and 113 days, respectively, after artificial infection through the scarified skin, contained worms and associated lesions in the liver, hepatic blood-vessels and other abdominal organs. Worms were found also in the kidney fat and ureter, under the capsule of the kidney, and in the pelvis of the kidney. It is important to note that worms were found immediately under the liver capsule, perforating the capsule, and in the body cavity. Two pigs, necropsied about 9 and 16 months, respectively, after experimental infection, showed for the most part healed liver lesions and worm infestations in the kidney region as well as in other organs.

The evidence obtained in this series of experiments shows that under experimental conditions which permitted skin penetration by the infective larvae of Stephanurus, the worms infected the liver and the hepatic blood-vessels as well as other abdominal organs, and also thoracic organs. The parasites appeared in the kidney regions rather late in the course of the infestation, and were absent from these areas as late as 99 days following experimental infection. That the worms which were found in the kidneys did not reach these organs through the circulation, as they did the liver, is shown quite conclusively by the results of the postmortem examination of the six pigs used in this series of experiments. The available evidence indicates that the larvae

which penetrated the skin followed a course of migration similar to that followed by the larvae which were swallowed. Following skin penetration, the larvae reached the blood-vessels of the liver, made their way from there into the hepatic tissue, and gradually migrated to the surface of the liver, ultimately perforating the liver capsule. Their transfer to the perirenal fat was accomplished by their active migrations in the abdominal cavity. The worms which reached the perirenal fat probably penetrated this relatively non-resistant tissue and reached the ureters through channels of communication established by the worm in the perirenal fat. Evidence to be published in another paper shows conclusively that Stephanurus perforates the ureter.

The fact that pigs killed as late as nine months after experimental infection contained worms which had not as yet attained the egg-laying stage, indicates that *Stephanurus dentatus* is a nematode which is unusually slow in developing. The fact that agamic worms get into locations in which they remain for a long time before moving on, many, in fact, failing to extricate themselves, may explain, at least in part, the slow course of development of these parasites.

3. EXPERIMENTS ON SUBCUTANEOUS INJECTIONS OF LARVAE

In order to obtain further data on the course of infection with *Stephanurus dentatus* in pigs which are exposed to the entry of the larvae through the skin, the following experiments were conducted:

Experiment V: On June 29, one-half cubic centimeter of a thick suspension of infective Stephanurus larvae was injected subcutaneously into each of four pigs. On September 12, or 75 days after experimental infection, the first pig (13) of this lot was killed. The postmortem observations are given below.

Liver with numerous darkened elevations which, on section, were found to contain dark caseous material and immature worms; connective tissue of liver greatly increased; surface of liver roughened; portal vein showed partially organized thrombi extending across the lumen of the blood-vessel; immature kidney worms present in the thrombi; no lesions found in gastro-hepatic artery; cysts in periportal tissue contained three immature worms. Fat surrounding kidney was free from kidney worms; kidneys showed white spots on superficial examination; no kidney worms present in these areas. One small thrombus was present in posterior vena cava, near its origin in liver; an immature kidney worm was found in this thrombus.

Another pig (14) from this lot was killed October 9, or 102 days after experimental infection. The postmortem observations are given below.

Surface of liver roughened and showing reddish linear lesions beneath capsule; numerous brownish elevations on liver; these elevations contained worms, some of which were escaping through ruptures in the liver capsule; perivascular connective tissue greatly increased and containing numerous pockets filled with pus and containing immature worms; lumen of portal vein and of gastro-hepatic artery almost occluded by thrombi which contained immature worms. Vena cava thrombotic.

A third pig (15) of this lot was killed October 16, 1928, or 107 days after experimental infection. The postmortem observations are given below.

Worms found in fat between stomach and liver along the course of portal vein; diaphragm and part of stomach wall roughened; one immature worm free on surface of stomach and one under the peritoneal coat of stomach; many worms free in abdominal cavity migrating on surfaces of abdominal organs. Liver with reddened linear lesions underneath capsule and with many nodules containing worms; several worms found perforating liver capsule; gastro-hepatic artery and portal vein thrombotic. Periportal connective tissue greatly thickened and containing many channels; worms found in fat between stomach and liver along course of portal vein; several worms found between hepatic tissue and hepatic lymph-gland, and in fatty tissue around pancreas. No worms in kidneys; 3 worms found in perirenal fat. Lungs normal; vena cava thrombotic, one worm present in thrombus.

The last pig (16) of this lot was killed December 20, 1928, or 174 days after experimental infection. The postmortem observations are given below.

Hepatic surface of stomach roughened; one kidney worm embedded in wall of stomach; several worms free in abdominal cavity between stomach and diaphragm. Surface of liver roughened and with many grayish elevations due to increased connective tissue; portal vein thrombotic and containing many immature kidney worms; lumen of gastro-hepatic artery almost occluded by thrombi; periportal connective tissue greatly increased and with many worms enclosed in pus pockets. Worms present in kidney fat but not in pus pockets; several worms in portion of kidney fat in direct contact with kidney; kidney shows

whitish scars beneath capsule, but no worms in parenchyma. Worms present in psoas muscle. Thoracic side of diaphragm with many wart-like prominences containing disintegrated kidney worms. Many worms present in wall of posterior vena cava, some of them in the process of perforating its wall; lungs with numerous pus pockets containing worms.

The data obtained in this series of experiments confirm those obtained in experiments on the penetration of the larvae through the scarified skin, and show that following subcutaneous injection the larvae go to the liver and ultimately perforate the liver capsule and reach the body cavity, from which they can readily get into the renal region.

Probable Course of Migration of Stephanurus in the Body of Swine

While we have not traced in detail the course of migration of Stephanurus larvae from the skin to the various thoracic and abdominal organs in which the parasites have been encountered in the course of the experiments which have been recorded in this paper, it seems safe to assume that the larvae reach the lungs through the circulation. The larvae can reach the right side of the heart either through the lymphatic system, by penetrating the lymphatic vessels and being carried in the lymphatic circulation to the thoracic duct, and thence to the left jugular vein and through the anterior vena cava to the right side of the heart, or they can be carried by the venous circulation directly to the right side of the heart. In experimental infection of guinea pigs, Stephanurus larvae have been found in considerable numbers in the intestinal lymph-nodes. From the right side of the heart the larvae would naturally be carried by the pulmonary arteries to the lungs. In these organs many of the larvae would be arrested, presumably, in the capillaries which might act as a sieve.

The transfer of the larvae from the lungs to the liver can be brought about as follows: These larvae which succeed in passing through the capillaries of the lungs would be returned to the left side of the heart by the pulmonary veins; from the heart the larvae would travel with the blood-stream in the dorsal aorta, and a number of them might get into the coeliac artery which divides into two main arteries, namely the gastro-hepatic and the splenic, and thus reach the liver and spleen. As noted in the protocols, the occurrence of the larvae in the gastro-hepatic artery and in the spleen has been demonstrated a number of

times in the course of the experiments on percutaneous infection of pigs with Stephanurus. The larvae can also reach the liver, by being carried with the systemic circulation to the intestine, where they can be transferred from the arterial capillaries to the venous capillaries and thus reach the portal vein. Stephanurus larvae appear to be especially attracted to the liver and in this organ many larvae become arrested and continue their development, ultimately getting out of the hepatic tissue by working their way to the capsule, which they perforate.

Larvae which reach the liver may continue with the blood-stream and this would bring them to the posterior vena cava in which blood-vessel they have been encountered in association with thrombi. While the larvae can and probably do reach the posterior vena cava through the circulation, it is not improbable that they may get into this blood-vessel by direct migration as a result of perforating the liver capsule, diaphragm and the wall of the vena cava. As a matter of fact, Stephanurus larvae exhibit striking tendencies to perforate various tissues as shown in connection with protocols given in this paper. Their occurrence in the periportal connective tissue may be due to their perforating the wall of the portal vein; on the other hand, the possibility that the larvae which reach the pancreas through the systemic circulation might migrate to the periportal connective tissue and from there into the portal vein by perforating the wall of this blood-vessel cannot be absolutely excluded.

That some larvae reach the kidneys through the systemic circulation is not improbable; the infarcts and scars observed in the kidneys of several pigs used in these experiments may have been caused by kidney worm larvae. As far as available evidence is concerned, larvae which might have succeeded in reaching the kidneys through the systemic circulation did not develop in these organs, as evidenced by the failure to find them there in the early stages of the life cycle, when they were abundant in the liver, lungs, pancreas, and in other organs. Worms which develop in the kidney region reach these areas relatively late in the course of their development, presumably as a result of an active migration from the liver and other locations to the perirenal fat.

Discussion

It is evident from the series of experiments which have been conducted with a view to determining whether the infective

larvae of *Stephanurus dentatus* are capable of penetrating the skin of swine, that infection with these parasites through the skin can be brought about under experimental conditions involving the scarification of the skin, or the subcutaneous injection of the larvae. Infection through the skin did not occur when viable larvae, obtained from cultures shown to be capable of producing infestations in pigs to which they were administered orally, were placed on the intact skin of young pigs.

Unpublished laboratory experiments designed to test the ability of the infective larvae to penetrate the skin of a young rat *in vitro* showed that these larvae were incapable of boring into intact, tender skin; known skin penetrators, such as the infective larvae of hookworms belonging to the genera Necator and Ancylostoma, have been shown to be able to penetrate the skin of young rodents, *in vitro*. However, in view of the readiness with which Stephanurus larvae penetrated the broken skin of pigs, with resultant worm infestations and associated lesions in various abdominal and thoracic organs and tissues, including the ultimate lodgment of the worms in the perirenal fat, ureter, and under the capsule and in the pelvis of the kidney, locations in which these parasites are known to attain fertile maturity, and from which their developing eggs can reach the outside with the urine, the question naturally arises as to the probable significance of the experimental data bearing on the penetration of Stephanurus larvae through the broken skin of pigs.

Considering the facts that swine are commonly exposed to conditions which result not only in skin abrasions, but also in cuts and other skin injuries, and that these animals are commonly infested with external parasites, particularly lice, which injure the skin, there can be no question but that infection with Stephanurus larvae through the skin is not only a theoretical possibility under normal conditions of swine husbandry, but is probably of common occurrence among swine raised in localities where kidney worm infestation is prevalent. Presumably the presence of sarcoptic mange mites in swine would be particularly favorable to the penetration of Stephanurus larvae because these parasites are known to destroy extensive skin areas, and this results in secondary cutaneous bacterial infection and further destruction of skin integrity. The damage to the skin by sarcoptic mange mites and other external parasites would offer opportunities for the penetration of Stephanurus larvae should the latter happen to come in contact with the damaged areas of the skin. The

opportunities for such contact would be particularly favorable in insanitary and permanent hog-lots, where kidney-worm eggs and larvae have ideal conditions for development and for survival for relatively long periods. Pigs, with skin abrasions and other skin injuries, lying down in such hog-lots, would become easy victims to the onslaught of infective larvae, which, activated by the warmth of the pigs' bodies, would be attracted to them, as they are attracted under laboratory conditions to a source of heat. This would result in their penetration through abraded or otherwise damaged skin.

Summary and Conclusions

The principal facts and implied conclusions brought out in this investigation may be summarized as follows:

1. When placed on the intact skin of pigs, the infective larvae of *Stephanurus dentatus* failed to produce infestations in the various tissues and organs in which these parasites are known to occur; the liver lesions observed in two out of six pigs which had been subjected to a normal percutaneous infection with kidney worm larvae are not considered as being due to these parasites. These findings modify the conclusions of Bernard and Bauche, who believed that percutaneous infection took place without reference to the integrity of the skin. Their success in percutaneous infection may have followed from the use of pigs with abraded skins.*

2. Infective Stephanurus larvae penetrated the scarified skin of pigs readily and produced typical lesions and worm infestations in the tissues and organs in which these parasites occur normally.

3. When injected subcutaneously into pigs, the larvae produced characteristic lesions and worm infestations in various tissues and organs which are susceptible to these parasites.

4. It is probable that under normal conditions of swine husbandry Stephanurus larvae would have opportunities of coming in contact with the injured skin of swine, and in such cases infestations with these parasites would result.

*In a recent paper on the kidney worm of pigs, by I. Clunies Ross, published in the *Journal of the Council for Scientific and Industrial Research*, iv (1931), pp. 30-33, which came to the attention of the writers while their manuscript was in the hands of the editor, the following statement occurs: "Infection of the host—the pig—may now be brought about with equal facility either through the larvae being swallowed by the pig, or through their active penetration of the animal's skin." This statement appears to be based on investigations carried out by Dr. Kauzal in Australia and is directly contrary to the writers' findings. The illustration given in the paper shows skin lesions on one side of the body which resulted, presumably, from the penetration of the larvae. As the skin on the sides of a pig's body is apt to be abraded or otherwise injured, it is not unlikely that the larvae in that case penetrated through the broken skin as they did in the cases reported in this paper.

5. Following experimental percutaneous infections, the larvae reached the liver, spleen, pancreas, perirenal fat, kidneys, psoas muscles and other abdominal organs and tissues; they also localized in thoracic organs, particularly in the lungs. These findings are not in agreement with the ideas of Bernard and Bauche, who thought that percutaneous infection resulted in perirenal infections but not in hepatic infection.

6. In experimental percutaneous infections the larvae were found in the liver and in adjoining abdominal organs and tissues long before they occurred in the perirenal fat and in the renal organs.

7. The worms apparently reached the liver through the systemic circulation and many of them remained in the hepatic blood-vessels, particularly in the portal vein and in the gastrohepatic artery, where they produced thrombi.

8. The immature worms penetrated the hepatic tissue, presumably by perforating the hepatic blood-vessels, and worked their way to the liver capsule which they perforated.

9. The worms which got into the abdominal cavity migrated freely and in this way they apparently reached the perirenal fat which they penetrated and in which they established tracts to the ureter.

10. That the worms reached the renal areas by an active migration was also evident from the fact that they appeared in that region relatively late in the course of experimental infections; they were absent in the kidney region in the earlier stages when the liver and other abdominal organs had been invaded. This was the case for infections both by the mouth and the skin, and this again is in disagreement with the findings of Bernard and Bauche. Liver involvement occurred in either case and was followed much later by renal involvement.

11. *Stephanurus dentatus* developed rather slowly in its swine host. This is probably due to the fact that the parasites had to go through various tissues and organs before they succeeded in reaching the renal region, which is the only location that affords an outlet for the eggs which the females produce.

12. In their migrations through the various tissues and organs many worms became encapsulated or otherwise arrested; such worms would apparently not succeed in completing their development to fertile maturity.

REFERENCES

[1]Bernard, P. N., and Bauche, J.: Influence du mode de pénétration, cutanée ou buccale, de *Stephanurus dentatus* sur les localisations de ce nématode dans l'organisme du porc et sur son evolution. Comp. rend. Acad. d. Sci., clvii (1913), 1, pp. 74-76.

[2]Bernard, P. N., and Bauche, J.: Influence du mode de pénétration, cutanée ou buccale du *Stephanurus dentatus* sur les localisations de ce nématode dans l'organisme du porc et sur son evolution. Ann. de l'Inst. Past., xxviii (1914), 5, pp. 450-469.

[3]Tayler, L.: Our present knowledge of the kidney worm (*Sclerostoma pinguicola*) of swine. 16th Ann. Rpt. B. A. I., U. S. Dept. Agr. (1900), pp. 612-637.

[4]Hellemans, J.: Ueber das Auftreten des *Strongylus pinguicola* Verrill, *Stephanurus dentatus* (Diesing) auf Java und Sumatra. Centralbl. f. Bakteriol. etc., Abt. I, lvii (1911), 3, pp. 212-239.

[5]Pecaud, M-G.: Supplément au Journal official de l'Africe occidentale francaise. Rapports et documents (1912). (This reference is not available to the writers.)

[6]Schwartz, B., and Price, E. W.: Observations on the life history of *Stephanurus dentatus*. (Abst. of paper Amer. Soc. Parasitologists.) Jour. Parasitol., xv (1928), 2, pp. 145-146.

[7]Schwartz, B., and Price, E. W: B. A. I. scientists make important discoveries on life history of kidney worm of swine. Official Rec., U. S. Dept. Agr., viii (1929), 35, p. 5.

[8]Schwartz, B., and Price, E. W.: Life history of the swine kidney worm. Science, n. s. lxx (1929), pp. 613-614.

[9]Schwartz, B.: Swine kidney worm causes loss to southern producers from condemned carcasses. Yearbook of Agriculture, U. S. Dept. Agr. (1931), pp. 499-502.

Convention Notes

Dr. C. C. Palmer, of Newark, Delaware, was the only veterinarian from his state at the convention. Dr. Palmer presented a paper before the Section on General Practice.

Dr. Adolph Eichhorn received a radiogram from the mid-Atlantic, sent by Dr. Thomas E. Smith, of Jersey City, N. J., who was on his way to Europe for a well-earned vacation.

All section officers were at the meeting except Dr. W. J. Butler, of Helena, Montana, chairman of the Section on Sanitary Science and Food Hygiene, who was kept at home on account of urgent official business.

Dr. Santiago Y. Rotea, of Manila, Philippine Islands, attended his first A. V. M. A. convention, although he has been a member for some time. He was very much impressed by the size and scope of the convention.

A telegram was received during the convention, from Dr. Benjamin McInnes, of Charleston, South Carolina, conveying best wishes for the success of the meeting and expressing his regrets at being unable to attend.

Dr. J. M. Armstrong, of East Providence, Rhode Island, and Dr. William H. Dodge, of Leominster, Massachusetts, represented the New England States. Dr. Armstrong used the air route in going from New York to Kansas City.

Dr. Clodoaldo A. Arias y Delgado, of Havana, Cuba, refuses to be discouraged about the A. V. M. A. meeting in Havana at some future date. With the 1932 convention awarded to Atlanta, Chicago a strong contender for 1933, and the 1934 meeting practically fixed for Philadelphia, 1935 seems to be the best bet for a visit of the A. V. M. A. to the Pearl of the Antilles.

NEWER KNOWLEDGE OF THE NEUROTROPIC VIRUS INFECTIONS OF THE HORSE*

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Studies which were undertaken in connection with an epizoötic of a peculiar malady which prevailed among horses and mules in the irrigated districts of the San Joaquin Valley during the summer and fall of 1930, and which became widely distributed in California and parts of Oregon in the summer of 1931, required a renewed critical analysis of the descriptions of the diseases referred to by veterinarians of this country as "cerebrospinal meningitis," "Borna disease," "non-purulent encephalitis," "forage poisoning," "Kansas-Nebraska horse plague," "botulism," etc. The task has been lightened by the excellent reviews of the older American literature in the papers by Udall,[1] Kinsley,[2] and Mohler.[3] Important data were found in a report of the Botulism Commission[4] published in 1922. For the purpose of comparison, numerous recent papers on these same subjects by Zwick,[5] Zwick and his associates,[6] Seifried,[7] Nicolau and Galloway,[8] Moussu and Marchand[9] and others have been consulted in the original texts.

Periodically many sections of the United States have been visited by a disease of horses the etiology and epizoölogy of which have baffled the best minds. Ever since Large[10] in 1847, Michener in 1850, Liautard[11] in 1869 and Williams[12] in 1897 reported in the East and in the West sporadic or group outbreaks of a febrile disease, which manifested itself in the form of a staggering, swaying gait followed by inability to rise, constipation, salivation, paresis and paraplegias involving the cranial nerves and a high mortality in from 1 to 4 days, it has been customary to diagnose such sickness as cerebrospinal meningitis or merely spinal meningitis. Doubtless the disease is enzoötic in many states. Single cases are probably not recognized or not properly diagnosed. In the past only epizoötics have attracted much attention. Thus, the outbreak of 1882, that in 1897 causing the loss of thousands of horses in Texas, the mortality of 600 horses

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in North Carolina in 1902, and those in Louisiana[13] in 1906 and 1907 have been recorded. In the course of an inquiry in California relative to the occurrence of past epizoötics the veterinarians frequently recalled small outbreaks in the communities in which they practice; thus it was reported that the same disease, which appeared in the San Joaquin Valley in 1930, prevailed in a less extensive way in the same districts in 1892. In 1919 and 1920, however, in this connection, it should be mentioned that according to Hart and Hayes[14] the cases which they have seen in the epizoötic in the Sacramento Valley in 1931 are clinically not identical in appearance with botulism as described by them in 1920.[15]

Outbreak of 1912 Widely Distributed

Probably the widest distribution of "cerebrospinal meningitis" was noted in 1912. Reports of cases were received by the Bureau of Animal Industry from Colorado, Georgia, Iowa, Kansas, Kentucky, Louisiana, Maryland, Missouri, Nebraska, New Jersey, North Carolina, Oregon, South Carolina, South Dakota, Virginia and West Virginia. Most disastrous was the disease in the central and western part of Kansas and Nebraska with an estimated equine mortality of about 35,000 head. The Botulism Commission (Geiger, Dickson and Meyer) attempted, in 1921, to collect further data and opinions from authoritative observers by sending a circular letter to all state veterinarians in the Union. Although the replies were non-committal and inconclusive they furnish ample evidence relative to the wide distribution and the magnitude of the losses which must in all probability be attributed to the same disease—"staggers" or "cerebrospinal meningitis"—even though it was, for reasons to be discussed, renamed "forage poisoning." It appears superfluous to repeat the details which are published (pages 72-75) in the report. However, since the Public Health Bulletin is not readily accessible, a few significant data are again mentioned.

An extensive outbreak of "forage poisoning," affecting 1,800 horses with a mortality of 1,000, occurred in Colorado during the months of July and September, 1919. Delaware, in 1919, lost approximately 100 horses in one county, while Montana reports 17 outbreaks, involving 1,004 horses and cattle; Nevada several outbreaks, with occasional reoccurrences on the same ranches; and South Dakota 1,500 horses, during the month of September, 1918. An approximate mortality of 3,000 horses has been

estimated for the five western states, California, Colorado, Oregon, Nevada and Montana, within the years 1915-1920. In recent years maladies of horses with symptoms of encephalitis have been seen in Kansas. In the state of Washington, Howarth[16] has seen sporadic cases in 1923-1928, with symptoms indistinguishable from those occurring in California in the epizoötic of 1930-1931. In California sporadic cases have been reported in past years resembling the encephalitis now prevalent. Unfortunately, the available statistical data are quite meager and consequently not subject to epizoölogical analysis. However, one generalization is probably permissible—the horse disease has definite tendencies to periodic, cyclical recurrences in extensive outbreaks. Aside from the seasonal fluctuations, epizoötics seem to appear at non-annual intervals of from 10 to 15 years.

The published clinical records (Kaupp,[17] Kinsley,[2] Mohler[3] and others) describe with rather monotonous uniformity a series of fairly characteristic symptoms and syndromes. Detailed postmortem descriptions stress the absence of any macroscopic lesions sufficiently marked to account for the intensity of symptoms. A careful perusal convinces the reader that in the majority of instances one and the same disease was recorded under different names. This is by no means surprising when one appreciates the lack of positive knowledge relative to the cause and nature of the disease.

So-Called Forage Poisoning

Despite the fact that in the large epizoötics it was impossible to connect the disease with any particular food, and competent observers had emphasized that climate, altitude and other epizoölogical facts excluded moldy hay or spoiled fodder, some authorities tenaciously have defended the diagnosis of "forage poisoning." The latter name unfortunately came into the literature as a synonym, in 1900, following the investigation by Pearson[18] of a disease among horses due to eating moldy silage. The malady was confined to 7 horses in a stable; 5 died after 2 to 4 days and 2 recovered. No autopsies were made. Two horses fed experimentally on the same lot of silage also died after showing tremors, paralysis of the pharynx and general paresis but no brain symptoms. Since clinically the disease of these horses resembled closely "sausage poisoning" in man, and since the toxic principle obviously resided in the food, Pearson suggested the name "forage poisoning." To the detriment of future progress, this

term has not been *restricted to sickness in animals in which it has been possible to establish an inseparable relationship between the feed containing a poisonous substance and the intoxication following its ingestion.* In fact, following the studies of Graham[19] and his associates, Buckley and Shippen,[20] Pammel,[21] Mitchell[22] and others, "forage poisoning" has quite generally been considered identical with botulism.

In the various reports it is stated that the symptoms and postmortem findings exhibited by the horses and donkeys experimentally poisoned with botulinus toxin resemble those generally noted in "cerebrospinal meningitis." The experiences of the writers, of Walker,[23] and of Theiler and his associates[24] on experimental equine botulism do not entirely support this contention. Symptoms of lethargy, sleepiness, loss of coördination resulting in staggering gait, muscular twitching, jaundiced and injected conjunctiva, accelerated pulse (60 to 120) and respiration rate *are not observed in botulism.* Paralysis of the tongue, one of the earliest and most characteristic signs of experimental equine botulism, is rarely if ever described as a prominent symptom in the epizoötic form of "cerebrospinal meningitis."

The postmortem findings are less conclusive, since reports on the microscopic examinations of the nervous system are limited to cases observed during the 1912 epizoötic. Typical vascular hemorrhages, perivascular infiltrations in the olfactory bulbs and pons as well as other signs of an encephalitis are mentioned by Udall,[1] Kinsley,[2] Mohler[3] and others. In experimental botulism these lesions are invariably absent.

So-Called Botulism

Aside from the clinical and anatomical differences enumerated between so-called "cerebrospinal meningitis" and "forage poisoning" it must be kept in mind that the occurrence of equine botulism is only suggested but not proven by Graham and others in four or five instances. The mere isolation of *Cl. botulinum* in enrichment cultures from the suspected feed is seriously invalidated by the established ubiquitous distribution of this anaerobe in the soil and on vegetation. The suspected botulinus toxin has not as yet been demonstrated in any of the feeds which have been associated with "forage poisoning." In fact, the senior author feels that the experimental evidence thus far published is inconclusive. As a working hypothesis equine botulism deserves further consideration, since Theiler and Robinson[25] have doubt-

less shown that stabled animals which may ingest foodstuffs contaminated by the decomposition products of dead animals may succumb to botulism. The South African strains were identified as type C.* It is not unlikely that in nature conditions suitable for the elaboration of the toxin may sometimes exist, but in extensive experimental studies conducted over several years the senior author has not succeeded in demonstrating the botulinus poison in feeds such as oats, corn, hay, grain, etc.

In this connection it is recalled that many of the early cases of human lethargic encephalitis showed symptoms of a paralytic nature very suggestive of botulism and were diagnosed as such in England during 1918.[26] In fact, equine "cerebrospinal meningitis," encephalitis or allied infections of the central nervous system, from a differential diagnostic standpoint, bear the same relation to "forage poisoning" and possibly botulism as human epidemic, lethargic encephalitis and cerebral lues bear to botulism in man.

Although the practicing veterinarian is frequently not in a position to observe and analyze the course of the symptoms, the lesions and the spread of the malady, he should, however, bear in mind that scattered cases of sickness, with nervous symptoms appearing in series among farm horses during the hottest months of the year, are most likely due to an infectious disease and probably not to botulism. The action of spoiled or moldy food or poisonous plants can usually be excluded on epizoölogical grounds.

So-Called Enzoötic Cerebro-Spinal Meningitis

In the Kansas-Nebraska outbreak of 1912, the feed showed, according to Udall,[1] no visible evidence of contamination or decomposition. The pastures were unusually dry; although Mohler stresses the fact that exceptional climatic conditions with high humidity prevailed in western Kansas. A careful and critical survey of the distribution, course and symptoms, and in particular the anatomical changes in the central nervous system led Udall to conclude that the equine disease of 1912 corresponded in all essential and fundamental features with the "enzoötic cerebrospinal meningitis" previously reported by Williams, from Idaho, and by Milks, of Louisiana. In fact, he even went a step further and definitely stated that the evidence at his disposal does not indicate that the disease is due to poisoning (forage poisoning in

*Horses are probably quite resistant to type C toxin.

the sense of Pearson) or that the food or water is a carrier of the toxic or infective agent.

Obviously Udall is inclined to consider the horse disease an infection and thus he identifies it with Borna disease, so ably studied in 1908 to 1910 by E. Joest,[27] Schmidt[28] and others in Saxony, Germany. This important and significant deduction has either been overlooked or has entirely been submerged by the prevailing theory of food contamination, and subsequently by the hypothesis of botulism. Several writers, impressed by the similarity of Borna disease in Germany and the American equine malady so prevalent in the New World, share the views of Udall, but since the cause of the German encephalitis was not known until 1925, they inclined toward the belief that toxic substances existing in the fodder generated by fungi or by bacteria were principally involved in the etiology of the sickness. Since case-to-case transmission had not been observed in America and the conception of unrecognized subclinical but immunizing infections were not as yet understood, the etiology of "forage poisoning" despite the very plausible theory continued to remain an unsolved problem.

Borna Disease not Found in America

In order to decide the identity or non-identity of the American equine malady with the German enzoötic encephalomyelitis or Borna disease, a brief review of the available facts appears necessary. The name Borna disease originated from the locality in Saxony where a severe epizoötic occurred in the years 1894 to 1896. The disease has been known for more than a century. In 1896, 1,198 horses were reported to have contracted the disease in several districts of Germany. Climatic conditions—abnormally warm and moist conditions—influenced the spread. As a rule, the sporadic cases reach an increased distribution in May and June, while relatively few instances of the disease are recorded during the fall and winter. (See diagrams published by Zwick, Seifried and Witte.[29]) The incubation time in the spontaneously contracted disease is difficult to estimate and is therefore not definitely known.

Experimental transmission experiments indicate that the incubation period may vary from several weeks to months. The symptoms, which draw attention to the infected animal, are lassitude and indifference to external, stimuli occasionally interrupted by periods of excitation, psychic and motoric disturbances,

difficulty in mastication and deglutition, contractions of diverse muscle groups, paresis and paralysis in the innervation centers of the cranial nerves, clamping of the jaws and excessive salivation, or the manifestations of encephalitis dominate the picture. In some cases the lesions are in the cord, while in others both brain and cord injuries induce the characteristic symptoms. As a rule, the temperature remains normal throughout or secondary rises occur as a result of septic complications. The examination of blood and urine does not indicate any constant change. The duration of the disease varies, according to the best authorities, from 1 to 3 weeks, in rare instances 4 to 6 weeks. The mortality is high, 80 to 90 per cent. Recovery is frequently incomplete and complicated by various sequelae (amaurosis, amblyopia, etc.). The gross postmortem examination reveals neither characteristic lesions in the central nervous system nor in the other organs.

Through the studies of Joest and Degen, important histologic changes were recognized in the olfactory bulbs, the nucleus caudatus, the hippocampus and the cord in the form of disseminated vascular round-cell infiltrations. These lesions are constantly present and are invariably accompanied by specific intranuclear inclusion bodies as reaction products of the ganglion cell toward the specific etiologic agent. As has been the case with a number of diseases subsequently proved to be due to a filtrable virus, the disease was at first attributed to the pathogenic action of various cocci. Streptococci and Gram-negative diplococci have been considered by the investigators concerned (Siedamgrotzky and Schlegel,[30] v. Ostertag,[31] Grimm,[32] Löhr,[33] Kraus[34] and others) as the etiological factor, until Zwick[5] and his associates succeeded, in 1925, in transmitting the infection to rabbits with brain emulsions of the diseased horses. Not only were transmissions with the infective material successful in dilutions of 1:100,000 but their detailed experiments proved that the encephalomyelitis is produced by a glycerin-resistant, ultravisible and filtrable virus.

Successive passages through rabbits, guinea pigs, rats and other animals, and reverse transmissions have been successful and the findings of Zwick and associates have been confirmed by Beck and Frohböse[35] and by Ernst and Hahn.[36] The infected laboratory animals manifest a disease which is clinically and anatomically identical with the spontaneous equine malady; cell inclusions are regularly found in the ganglion cells of the hippocampus and other structures. (See Nicolau and Galloway,[38] Cowdry.[37]) Aside from the intracerebral route of transmission, which is experi-

mentally the most successful, intralumbar, intra-ocular, corneal, intravenous, intranasal and repeated subcutaneous administration of the virus may produce the typical disease in rabbits.

Of interest is the fact that the incubation time of the experimental infection varies from 2 to 6 weeks and the duration 10 to 14 days; in the horse, 4 to 8 weeks. It is therefore not unlikely that in the spontaneous cases a definite latent period of several months elapses between the invasion of the virus and the appearance of the first symptoms. According to recent investigations of Nicolau and Galloway,[38] the generalization of the virus throughout the body is well proven. The transmission of the disease under natural conditions is probably by the respiratory tract (nasal passages) or by ingestion. The saliva and the nasal secretions contain the virus. Zwick and his associates, in a series of mass exposure experiments, demonstrated that the infection may be transmitted by cohabitation, from sick to healthy rabbits and rats. Repeated subcutaneous injections of virulent brain suspensions render horses non-susceptible to subsequent intracerebral test inoculations of the virus. The serum of the immune horses contains neutralizing antiviral substance. Such sera have been used therapeutically without any noteworthy success.

A Second Type Reported in Germany

The data just presented refer to the form of encephalomyelitis as observed and studied in Germany and called Borna disease. Recent publications indicate that a second type, described by Fröhner[39] and by Dobberstein[40] in Berlin, deserves consideration in the future. The comparative microscopic examination of the brains revealed a non-purulent hemorrhagic encephalitis with particular localization of the lesions in the medulla, pons, cerebellum and lumbar cord. Transmission experiments have not been attempted. In fact, the etiology is unknown. In this connection, one recalls the reports of MacCallum and Buckley[41] on an acute hemorrhagic encephalitis and the descriptions of Tait Butler[42] on a malady designated by him as leuco-encephalitis. Butler reports four cases of this kind followed eating moldy corn in Kansas and about 600 cases in an outbreak in North Carolina. He emphasizes the fact that the disease differs from "cerebrospinal meningitis" and "forage poisoning" of Pearson. It is obviously impossible to state whether the Fröhner-Dobberstein encephalomyelitis is in any way related to the Butler type of equine encephalitis. In future investigations, however, the possibility should be kept in mind.

Shortly before Zwick and Seifried published the experimental transmission of the German Borna virus to small laboratory animals, Moussu and Marchand[43] announced (1922) and reported later (1924) in a monograph a detailed histologic and etiologic study on l'encéphalite enzootique du cheval, as seen in France. The observations were made in May and June, 1922, in a stud of pure-bred horses. In a group of 80 mares, foals, yearlings and two stallions, 15 cases with six deaths were noted during a period of two months. Clinically the French workers observed three forms: the cerebral, the spinal and the mixed. Of particular interest is the fact that the course of the disease was quite acute. The animals succumbed to the cerebral form within 20 to 37 hours and to the mixed form within six days.

The autopsy reports record an immediate onset of the postmortem decomposition, a splenic tumor and the general picture of a rapidly progressing septicemia with parenchymatous changes in the liver and kidneys. The macroscopic alterations in the central nervous system were slight and confined to stasis in the blood-vessels and petechiae on the meninges. The lesions are microscopically indicative of an encephalitis although they are confined to hemorrhages around the capillaries of the medulla and to tigrolysis and hyperchromatosis of the ganglion cells. A search for inclusion bodies has been entirely unsuccessful.

Moussu and Marchand reproduced the disease by inoculation of virulent brain suspensions from the horse into the anterior chamber of the eye of rabbits. It is of interest that, in one experiment, of 8 rabbits inoculated only 2 became infected. The incubation time varied between a few days to several weeks. On repeated passage from rabbit to rabbit the virus became fixed and caused fatal infections in 3½ to 6½ days. The course was acute and primarily governed by excitation and motoric disturbances. Reverse passages from the rabbit to the horse led to a fatal encephalitis in 3 days. Transmission in series rabbit-horse-rabbit was successful. Of particular interest is the fact that guinea pigs, rats and mice proved refractory when the virus was administered by the intra-ocular route. Feeding, subcutaneous and nerve inoculations were tried. The virus was found to be ultravisible and filtrable; it preserved its virulence in glycerin at room temperature for 18 days, but it lost its pathogenic action after 32 days.

Moussu and Marchand concluded from their studies that the equine encephalitis as observed by them in France was identical

with Borna disease. This deduction, until recently shared by Nicolau and Galloway, is by no means justified, for reasons which have been presented by Zwick[5] in an impartial but critical analysis of the essential facts. He emphasizes the prominent involvement of the spinal cord, the absence of inclusion bodies, the presence of hemorrhages, the short incubation time and the acute course of the experimental disease as outstanding differences which are not observed in the infection known as Borna disease. In fact, he concluded that the epizoötic encephalitis (Moussu and Marchand) is an acute, severe, infectious disease of septicemic character with predominant involvement of the central nervous system, while Borna disease is an elective neurotropic infection of the brain and cord without any particular participation of the body as a whole. This conception is supported by Seifried,[7] who therefore places the Moussu-Marchand disease into the second group of the encephalomyelitis with organotropic properties of the virus.

A Disease Reported from Argentina

Before consideration is given to the encephalitis seen on the North American Continent it may be appropriate to inquire briefly into the nature of an epizoötic equine disease which has been studied by Quevedo, Kraus, Kantor and Quirego,[44] in Argentina, in 1919. The incomplete clinical and anatomical descriptions suggest a form of meningo-encephalitis without intranuclear inclusions in the ganglion cells. Kraus reports the successful isolation of a Gram-negative, coffee-bean-shaped diplococcus with which he reproduced the infection by intracerebral injection in the horse, by subdural and intravenous administration in calves, sheep and goats, and by the intravenous and subcutaneous routes in rabbits, guinea pigs and mice. Despite the fact that the clinical picture and the course of the infection as produced in the rabbits differed strictly from that induced by the Borna virus, he continued to defend, until 1925, the conception that the Argentinian enzoötic encephalitis is identical with Borna disease and caused by a readily cultivated specific diplococcus. A critical analysis fails to support this view and one is forced to conclude that the etiology of the disease in Argentina remains undetermined.

Encephalomyelitis in California

The epizoötic encephalomyelitis which prevailed in the San Joaquin Valley, during the month of July and until November,

1930, affected approximately 6,000 horses and mules with a mortality of 50 per cent. In July, 1931, the disease reappeared in both the Sacramento and San Joaquin valleys and in various other parts of California. It is an infectious disease with an incubation time of from 1 to 3 weeks. Preceding the onset of the symptoms which attract attention, the temperature may be found to vary from 103 to 107° F. Not infrequently when the horse shows signs of drooping of the head, sleepiness and circling motion or other psychic and motoric disturbances, the body temperature may be normal. The pulse and respiration are usually accelerated. Quite often the animal rests against the wall or corner and may show backward and sideways motions. Muscular twitchings are quite common.

Many of the horses are down on the second or third day and may or may not get up when pressed to do so. Paresis of the lips and drooling is frequently noted. Mastication and swallowing may or may not be impaired but grinding of the teeth is quite regularly observed. The conjunctiva is always injected and frequently icteric or grayish and studded with petechiae or ecchymoses. In the mild cases which were able to rise, recovery was as a rule uneventful but about half were so severe that they terminated fatally in 3 to 8 days or were destroyed for humane reasons.

Gross Anatomical Lesions Absent

Aside from a slight general icterus, moderately succulent lymph-nodes and parenchymatous changes in the liver and kidneys, no gross anatomical lesions were found at autopsy. The spleen was not enlarged. The spinal fluid was slightly increased and showed 12 to 30 cells per cmm., mostly lymphocytes and a few leucocytes. As a rule, the brain and cord were moist and soft irrespective of the advanced dehydration which was present in several cases. The most obvious and striking microscopic changes in the brain consisted of hemorrhages around the vessels of the olfactory bulbs and brain stem. Infiltration of the perivacsular sheaths and spaces due to mononuclear and polymorphonuclear cells was variable in intensity. This cuffing of the veins and arteries was definite in the advanced cases of the disease. Scattered patches of infiltration in the gray, and occasionally in the white, matter were common. The distribution and the intensity of the inflammatory foci differ from those commonly seen in typical Borna disease. Nuclear inclusion of the character described as typical

of Borna disease by Joest and Degen has not been observed in the material thus far examined.

The clinical and anatomical findings were in nearly every respect identical with those previously reported by observers of the American form of encephalitis popularly known as "forage poisoning" or "cerebrospinal meningitis."

Transmissibility Experiments

Experimental studies in California, to be reported elsewhere, have proved conclusively the transmissibility of the infective virus, which is present in the central nervous system, to horses, monkeys, rabbits, guinea pigs, rats and mice by subdural or intracerebral injections of brain suspensions. Cultures from field cases in horses, prepared from the spinal fluid and occasionally the brain, gave both hemolytic and non-hemolytic streptococci which were non-pathogenic and without etiologic significance. The most suitable laboratory animal for the study of the filtrable, glycerin-resistant virus is the guinea pig. Not only intracerebral injections but intranasal instillations of brain emulsion transmit the virus to those rodents, causing an acute fatal disease with death in from 4 to 6 days. Reverse passage experiments through guinea pig, horse, rabbit, guinea pig, horse and monkey have as a rule been successful even with filtered and diluted (1:1000) virus suspensions. Rabbits are readily immunized; their serum neutralizes the virus when mixed *in vitro* with the serum and thus tested by intracerebral injection on guinea pigs. The detailed comparative investigations are still in progress.

Early in the course of the study of the San Joaquin Valley disease, it became evident that the infection is identical with that previously observed on the American Continent. Based on microscopic examinations of the brain and without previous clinical examinations of the affected horses the tentative diagnosis of Borna disease was made by the writers in September, 1930. Preliminary cross-immunization tests now indicate that the American virus is not identical with the Borna virus. With the accumulation of epizoölogical and other pertinent data it has become evident that the disease resembles the Moussu-Marchand type of epizoötic encephalitis. Unfortunately a comparison of the French with the American virus is not feasible since the French strain is not available. The American infection shares with the French form the acuteness of the spontaneous

and experimental disease but it differs in a number of characteristics which are probably not merely expressions of degree. Septicemic lesions with splenic tumors have not been noted in the American disease. Moreover, the blood at the height of the febrile reaction in the latter disease has not been found to be infectious.

Rabbits are not very susceptible to the French virus. A fatal disease is regularly produced when the virus has been adapted by passage. Clinically recognizable infections in the rabbit with the American virus are rare but when the animal is sacrificed at the height of the febrile reaction the presence of the American virus can be demonstrated by subinoculation on the susceptible guinea pigs. Then again the French virus has been transmitted only to the horse and rabbit on intra-ocular injection, while the American virus infects by the cerebral, ocular and nasal routes and is capable of inciting encephalitis in guinea pigs, monkeys, rats and mice.

A comparison of the clinical and anatomical changes indicates fundamental differences which have already been discussed. Then within the scope of the available observations it is noteworthy that the American epizoötics began in Kansas and in California in July or early August and reached their maximum intensity in September, only to disappear after the coming of frosts or cool nights in October or early November. The incidence is obviously the highest in the hottest months of the year. In this respect, the equine malady shows a behavior analogous to human infantile paralysis. The rare French epizoötics—so far only two reported—occurred during the month of May among the stabled horses of a stud. Farm horses and mules are chiefly affected in the United States. The mode of transmission and spread in California is as yet unknown. A few scattered instances of contact infection have been noted but as a rule conveyance of the infectious agent from animal to animal accompanied by clinical symptoms is apparently rare. Until the nature of the immunity or the predisposing factors of susceptibility are understood, no explanation can be given for these epizoölogical facts. Intensive field studies quite essential for the elucidation of such problems are seriously handicapped, since an adequate system of reporting communicable disease of animals is rarely available. The veterinary "epidemiologist" is therefore largely dependent on speculation and analogy deductions.

The evidence thus far obtained in California indicates that the American encephalitis differs etiologically from both the Moussu-Marchand type and also Borna disease. Until certain further investigations are completed it must remain undecided whether the American disease is due to an elective neurotropic virus or whether it is an encephalomyelitis with organotropic properties of the virus, as for example is the case in hog cholera, distemper, fowl pest and yellow fever.

References

[1]Udall, D. H.: A report on the outbreak of "cerebro-spinal meningitis" (encephalitis) in horses in Kansas and Nebraska in 1912. Cor. Vet., iii (1913), 1, pp. 17-43.

[2]Kinsley, A. T.: Forage poisoning or equine encephalomyelitis. Proc. 50th Ann. Meet. A. V. M. A. (1913), pp. 413-426.

[3]Mohler, J. R.: Forage poisoning or cerebro-spinal meningitis. Amer. Vet. Rev., xlii (1913), 5, pp. 506-522.

[4]Pub. Health Bul. 127 (1922), p. 69.

[5]Zwick: Berl. tierärzt. Wchnschr., xli (1925), p. 453; Seuchenbekampfung, iii (1926), p. 57.

[6]Zwick et al: Ztschr. f. Infekt., xxx (1926), p. 43; xxxii (1927), p. 150; Arch. f. wissensch. u. prakt. Tierh., lix (1929), p. 511; Handb. der pathog. Mikroorg., ix (1927), p. 117.

[7]Seifried, O.: Ztschr. f. d. ges. Neurol. u. Psychiat., cxxiv (1930), p. 317; Ergebn. d. allg. Path. u. Anat., xxiv (1931), p. 534.

[8]Nicolau and Galloway: Medical Research Council Special Report, 121 (1928); Ann. de l'Inst. Past., xliii (1929), p. 1; xliv (1930), p. 673; Compt. rend. Soc. de biol., ciii (1930), p. 852.

[9]Moussu and Marchand: L'encéphalite enzootique du cheval (Vigot Frères, Paris, 1924).

[10]Large: Veterinarian, xl (1867), p. 655.

[11]Liautard, A.: Rec. de méd. vét., xlv (1869), p. 361.

[12]Williams, W. L.: Enzoötic cerebro-spinal meningitis in horses, and hog cholera in Idaho. 14th Ann. Rpt. U. S. B. A. I. (1898), pp. 179-187.

[13]Milks, H. J.: La. Agr. Exp. Sta. Bul. 106 (July, 1908).

[14]Hart, G. H. and Hayes, F. M.: Personal communication (1931).

[15]Hart, G. H. and Hayes, F. M.: The production and experimental use of botulinus antitoxin, types "A" and "B." Jour. A. V. M. A., lvii (1920), n. s. 10 (6), pp. 638-652.

[16]Howarth, J. A.: Personal communication (1931).

[17]Kaupp, B. F.: Cerebrospinal meningitis of the horse. Proc. 50th Ann. Meet. A. V. M. A. (1913), pp. 402-412.

[18]Pearson, L.: A preliminary report upon forage poisoning of horses (so-called cerebro-spinal meningitis). Jour. Comp. Med. & Vet. Arch., xxi (1900), pp. 654-657.

[19]Graham et al: Ky. Agr. Exp. Sta. Buls. 207 and 208 (1917); Univ. of Ill. Ext. Cir. 38 (June, 1920); Jour. Bact., iv (1919), p. 1; vi (1921), p. 69.

[20]Buckley, J. S. and Shippen, L. P.: Preliminary report on the relation of anaerobic organisms to forage poisoning. Jour. A. V. M. A., 1 (1917)), n. s. 3 (7), 809-816.

[21]Pammel, L. H.: Am. Jour. Vet. Med., xiii (1918), p. 3.

[22]Mitchell, C. A.: Preliminary report on equine botulism in Canada. Jour. A. V. M. A., lx (1922), n. s. 14 (5), pp. 609-619.

[23]Walker: Brit. Jour. Exp. Path., x (1929), p. 352.

[24]Theiler, A., et al: 13th and 14th Rpts. Dir. Vet. Educ. & Res., Union of South Africa, Part 1 (Oct., 1928), p. 47; Rev. gen. de med. vet., xxxvi (1927), p. 193; So. Afr. Jour. Sci., xxiii (1926), p. 526.

[25]Theiler and Robinson: Ztschr. f. Infekt., xxxi (1927), p. 165.

[26]Rpt. Loc. Gov. Board on Pub. Health & Med. Subjects, n. s. 121 (London, 1918).

[27]Joest, E.: Ztschr. f. Infekt., ix (1911), p. 1.

[28]Schmidt: Mag. Tierheilk. (1870), p. 186.

[29]Zwick, Seifried and Witte: Ztschr. f. Infekt., xxx (1926), p. 43.

[30]Siedamgrotsky and Schlegel: Arch. f. wissensch. u. prakt. Tierh., xxii (1896), p. 287.

[31]v. Ostertag: Berl. tierärztl. Wchnschr., xvi (1900), p. 433; xxii (1906), p. 622.

[32]Grimm: Berl. tierärztl. Wchnschr., xliii (1927), p. 153.

[33]Löhr: Thesis, Dresden (Leipzig, 1910).

[34]Kraus: Ztschr. f. Immununitätsforsch. u. exp. Therap., xxx (1920), p. 121.

[35]Beck and Frohböse: Arch. f. wissensch. u. prakt. Tierh., liv (1926), p. 84.

[36]Ernst and Hahn: München. tierärztl. Wchnschr., lxxvii (1926), p. 46.

[37]Cowdry: Science, lxviii (1928) p. 40.

[38]Nicolau and Galloway: Ann. de l'inst. Past., xliv (1930), p. 673.

[39]Fröhner: Berl. tierärztl. Wchnschr., xl (1924), p. 477.

[40]Dobberstein: Ztschr. f. Infekt., xxxiii (1928), p. 290.

[41]MacCallum and Buckley: Jour. Exp. Med., vi (1905), 1, p. 65.

[42]Butler, T.: Notes on a feeding experiment to produce leucoencephalitis in a horse, with positive results. Am. Vet. Rev., xxvi (1902), 8, pp. 748-751.

[43]Moussu and Marchand: Rec. de méd. vét., xcviii (1922), p. 261.

[44]Queredo, Kraus, Kantor and Quirego: Rev. Inst. Bact. Buenos Aires (1919); Berl. tierärztl. Wchnschr., xli (1925), p. 268.

UNICORNUAL UTERUS IN A GUERNSEY HEIFER

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This report concerns a case of sterility in a two-year-old Guernsey heifer, which apparently was due to a malformation of the uterus.

This heifer (not a twin) had a history of never coming in heat except a few times when 6 to 9 months of age. An examination

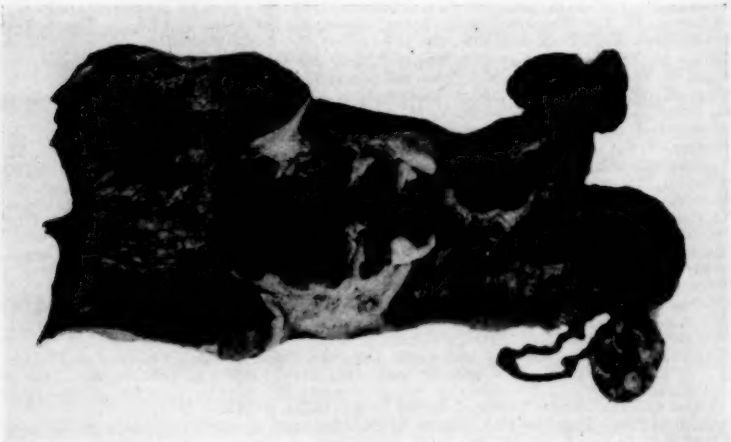

FIG. 1. Interior of the genital tract in case of unicornual uterus in heifer.

was made and only the right horn and ovary could be palpated. The latter contained a large corpus luteum which was expressed. As this heifer was extremely nervous, it was decided not to examine her further at that time.

The herdsman was instructed to watch this animal carefully for evidence of estrum. After a period of two months following the examination, he reported she had not come in heat.

A second examination was made, with similar results as the first. Upon further search, a small, flat, tense, fibrous band was found which replaced the left horn. The left ovary was palpated and found to be attached closely to the end of this band. It also contained a large corpus luetum. As a further check, this heifer was examined again three days later and the findings were the same. She was then sent to the packing-house, where her genital tract was removed for examination.

Figure 1 shows the interior of the genital tract. The mucosae of the vagina, cervix and right horn are easily recognized; also the right ovary and tube. The small, dense, fibrous band, with an incision in it, is seen replacing the left horn. The left ovary, cut through the center and exposing the corpus luteum, is seen closely attached to this dense fibrous band. No fallopian tube could be found.

THE THERAPEUTIC VALUE OF BRUCELLA ABORTUS BACTERIN ADMINISTERED INTRAVENOUSLY*

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On different occasions practitioners have requested information on the value of *Brucella abortus* (Bang) bacterin, administered intravenously to reacting cows. Claims are sometimes made that animals which react to the agglutination test will become negative in response to repeated intravenous injections of bacterins. Controlled experimental data in support of this contention have not been presented. In order to assist clinicians interested in the above hypothesis, ten cows that had reacted for varying periods of time, dating back to April, 1925, were injected intravenously in October, 1930, with 2-cc, 3-cc and 4-cc doses of *Brucella abortus* (Bang) bacterin at weekly intervals. Agglutination tests on the bacterin-injected animals were conducted for six consecutive months following treatment. Both blood and milk were tested by the standard tube method. (See table I.)

Summary: The intravenous injection of cattle reacting to Bang's disease with killed Brucella cultures (porcine strain) failed to alter appreciably the agglutinin content of blood and milk of ten animals over a period of six months following treatment. No ill effect of treatment was noted.

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TABLE I—*Brucella abortus bacterin administered intravenously to reacting cows*

Cow	Breed	History		Agglutination 10–14–30		Agglutination of Blood and Milk After Treatment*													
						11–11–30		12–1–30		1–1–31		2–1–31		3–1–31		4–1–31		5–1–31	
		Born	Reacted	Blood	Milk	B	M	B	M	B	M	B	M	B	M	B	M	B	M
294	Ayr.	12–26–29	Apr. '25	1:200	N	1:200	1:200	1:200	N	1:200	Dry	1:200	N	1:200	N	1:200	N	1:200	N
364	Hols.	1–23–24	Aug. '26	1:200	1:200	1:200	1:200	1:200	1:200	1:200	N	1:200	1:200	1:200	1:200	1:200	1:200	1:200	1:200
374	Br. Sw.	3–20–19	Nov. '27	1:200	1:50	1:200	1:200	1:200	1:200	1:200	Dry	1:200	N	1:50	N	1:200	1:200	1:200	1:50
319†	Ayr.	3–20–21	Apr. '25	1:200	Dry	1:200	1:200	1:200	1:200	1:200	Dry	1:200	1:200	1:200	N	1:200	N	1:200	1:200
393	Br. Sw.	4–30–19	Dec. '27	1:200	1:50	1:200	1:200	1:200	1:200	1:200	N	1:200	N	1:200	N	1:200	N	1:200	N
420†	Hols.	11–25–26	July '28	1:200	Dry	1:200	1:200	1:200	1:200	1:200	Dry	1:200	Dry	1:200	Dry	1:200	N	1:200	1:200
300	Guer.	9–30–20	Apr. '25	1:200	1:50	1:200	1:200	1:200	N	1:200	N	1:200	N	1:200	1:50	1:200	1:50	1:200	N
369	Guer.	2–24–24	Apr. '25	1:200	1:50	1:200	N	1:200	N	1:200	N	1:200	1:200	1:200	N	1:200	N	1:200	N
352	Jer.	9–29–23	Dec. '25	1:200	1:200	1:200	1:200	1:200	1:200	1:200	1:200	1:200	Dry	1:200	1:50	1:200	1:50	1:200	N
372	Jer.	3– 4–25	Apr. '26	1:200	N	1:200	1:200	1:200	1:200	1:200	1:200	1:200	Dry	1:200	N	1:200	1:200	1:200	1:200

*Treatment consisted of three intravenous injections of killed culture of *Brucella suis* (Traum)—density of the McFarland Nephelometer No. 1. 10-14-30, 2 cc; 10-21-30, 3 cc; 10-28-30, 4 cc.

†Animals not in milk. The udder secretion, upon manipulation, proved varied in quantity.

ORNITHOSTRONGYLUS QUADRIRADIATUS IN SQUABS*

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In 1904, Stevenson[1] investigated a malady which destroyed the greater portion of a flock of pigeons in Washington, D. C., and recovered from the small intestine a filiform coiled nematode, which he fully described and named *Strongylus quadriradiatus*. Since then, the parasite has been recorded by Cram,[2] who used the name *Ornithostrongylus quadriradiatus*. Because of the scarcity of reports concerning the distribution of this pathogenic parasite, it was thought that it might be of value to record the following case:

On June 23, 1931, two squabs were presented for examination by Mr. P., from Paterson, N. J. On June 30, two additional squabs were received from the same source. The owner had a flock of about fifty pigeons and lost all but six of them in a comparatively short time. The squabs from the second lot presented the following, on postmortem examination:

Condition, poor; mucous membranes, pale; feathers around anus, soiled. The lungs were somewhat congested; other parenchymatous organs appeared to be normal. The mucous membrane of the duodenum and small intestine was found to be swollen, roughened, hemorrhagic in spots, and covered with a thick tenacious somewhat granular exudate. The number of parasites was enormous in the duodenal region, and somewhat less in the terminal portion of the intestine. A tentative diagnosis of *O. quadriradiatus* was made, which was later substantiated by a careful microscopic examination.

DIAGNOSIS

The diagnostic features of this nematode, as given by Stevenson,[1] and which can be readily recognized in a fresh, mounted specimen under the microscope, are as follows:

Male: 9-12 mm. long, filiform, coiled, red in color. Cephalic end blunt, bursae bilobed, six rays arranged in pairs in each lobe. Two three-pointed spicules joined by membrane to form a tube, and supported by a characteristic star-shaped, chitinous piece.

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Pathological Significance

O. quadriradiatus is a blood-sucking parasite, its red color being due to engorgement with blood.[2] The position of the parasites in the mucosa of the intestinal canal causes severe irritation, as indicated by copious exudate, and the persistent diarrhea. Loss of blood through hemorrhages into the lumen of the intestine, and through sucking causes a noticeable anemia. The presence of the parasite in great numbers proved to be fatal.

Summary

The etiological agent of an epizootic of pigeons, in which all but six succumbed, was found to be a nematode worm identified as *Ornithostrongylus quadriradiatus.*

References

[1]Stevenson, E. C.: A New Parasite (*Strongylus quadriradiatus* n. sp.) found in Pigeons. U. S. D. A., B. A. I. Circular 47 (June 30, 1904), pp. 1-6.
[2]Cram, E. B.: Nematodes of Pathological Significance Found in Some Economically Important Birds in North America. U. S. D. A. Tech. Bul. 49 (January, 1928), pp. 4-5.

Addendum

Since the above report was written we had occasion to encounter two additional infestations with this parasite.

On July 14, two pigeons were presented from Sayreville, Middlesex County, New Jersey, with the history that since July 10, twenty pigeons had died in a flock of about 100, of which 6 were adults.

The second case came on July 15, from New Brunswick, Middlesex County, New Jersey. All of a loft of seventeen pigeons were said to have died in a few days.

In both cases, the intestines were catarrhal, hemorrhagic and very heavily infested with the parasite.

THE EFFECT OF COLLOIDAL CARBON WITH ADSORBED FLAVINES ON THE BRUCELLA AGGLUTININ TITRE OF REACTING COWS*

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Colloidal carbon and flavine dyes in combination, according to propounded theory, possess bactericidal properties for *Brucella abortus* (Bang), in the tissues of infected animals, not possessed

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TABLE I—*The effect of colloidal carbon with adsorbed flavines on the Brucella agglutinin titre of reacting cows*

SPECIMEN	HISTORY OF REACTIONS 1:200		TREATMENT	AGGLUTINATION OF BLOOD AND MILK													
				8–18–30		8–25–30		9–1–30		9–8–30		9–15–30		9–22–30		3–22–31	
	BLOOD	MILK		B	M	B	M	B	M	B	M	B	M	B	M	B	M
2839	Oct. '28 Aug. '29 Sep. '29 Oct. '29 Apr. '30	Sep. '29 Oct. '29 Apr. '29	Colloidal Carbon with Absorbed Flavines—50 cc intravenously 8–18–30 8–25–30 9– 1–30 9– 8–30 9–15–30 9–22–30	1:200	N	1-200	1:50	1:200	N	1:200	1:50	1:50	N	1:200	N	1:200	N
2840	May '29 Jun. '29 Sep. '29 Oct. '29 Feb. '30 Apr. '30	May '29 Sep. '29 Oct. '29 Feb. '29 Apr. '19		1:200	1:200	1:200	1:200	1:200	1:200	1:200	1:200	1:200	:1200	1:200	1:200	1:200	1:200
2841	May '29 Aug. '29 Oct. '29 Feb. '30 Apr. '30	Oct. '29 Apr. '30		1:200	N	1:200	1:50	1:200	N	1:200	N	1:200	N	1-200	N	1:200	1:200

by either substance alone. In an attempt to cure Bang's disease, practitioners have injected, on different occasions, colloidal carbon with adsorbed flavines. This combination has been recommended in the treatment of deep-seated bacterial infections, with the hope of stimulating phagocytosis, and at the same time favoring the bactericidal effect of the flavines upon the invading microörganism. In previous experiments,[1] flavines as well as colloidal carbon have been employed separately in treating infected cows, with negative results. The value of the treatment was arbitrarily measured by repeated application of the agglutination test to treated animals. The effect of intravenous medication was observed on three animals that had proven, on previous occasions, to be positive to the agglutination test. (See table I.)

Summary: The results of weekly blood tests during the course of the treatment and after an interval of approximately six months failed to indicate the curative properties of colloidal carbon with adsorbed flavines in the treatment of reacting animals. No injury as the result of treatment was observed.

REFERENCE

[1]Graham, Robert, and Thorp, Frank, Jr.: Studies in Chemotherapy of Bang's Disease (Bovine Infectious Abortion). Corn. Vet., xx, (1930), 4, pp. 325-344.

Convention Notes

Outbreaks of anthrax and hog cholera in various parts of Iowa kept down the attendance of Hawkeye veterinarians.

The University of Minnesota was represented by six veterinarians: Drs. C. P. Fitch, W. L. Boyd, H. C. H. Kernkamp, C. R. Donham, R. Fenstermacher and W. A. Billings.

Although the attendance figure reached approximately 1450, it was barely possible to round up 150 railroad certificates. The goal was reached, however, and those who traveled by rail enjoyed the benefit of the reduced fare for the return trip.

Official Washington was represented by Drs. John R. Mohler, A. E. Wight, Maurice C. Hall and George W. Pope, of the Bureau of Animal Industry; Harry E. Moskey, of the Food and Drug Administration; and J. E. Shillinger, of the Bureau of Biological Survey.

Among the old-timers in attendance were Drs. E. B. Ackerman (1891), C. A. Cary (1890), J. W. Connaway (1890), W. F. Crewe (1893), W. H. Dodge (1892), G. A. Johnson (1890), Wm. Henry Kelly (1896), N. S. Mayo (1891) and L. A. Merillat (1893). (The dates in parentheses indicate the year in which each of these members joined the A. V. M. A.)

THERMAL DEATH POINT. I. Spores of *Clostridium tetani*. T. J. Murray and Mary R. Headlee. Jour. Inf. Dis., xlviii (1931), 5, p. 436.

The spores of *Cl. tetani* in physiologic sodium chlorid solution were killed at 105° C., the time varying between 5 and 10 minutes; at 100° C. between 10 and 25 minutes and at 95° C. between 25 and 60 minutes. At 95° C. three strains resisted the 60-minute heating. Dry spores heated in the absence of moisture were killed at 140° C., the time varying from 5 to 15 minutes; at 135° C., from 5 to 25 minutes; and at 125° C., from 20 to 40 minutes. Dry spores heated under moist conditions showed no increased resistance, such spores being killed at 105, 100 and 95° C. In many of the strains drying decreased the thermal resistance. Drying of the spores of *Cl. tetani* on sand decreases the number of viable spores and also tended to decrease their resistance when heated under moist conditions. Spores of *Cl. tetani* showed best resistance at pH 7. Acid reactions were more toxic than alkali reactions. Increasing amounts of organic matter (peptone) tended to increase the thermal death time when the suspending solution was at pH 7. Above and below this, at pH 10 and pH 5, the effect mentioned did not occur. Spores of *Cl. tetani* suspended in a solution of sodium chlorid showed the greatest resistance to heat at a salt concentration of 2 per cent. The authors conclude that two factors were involved in the killing of spores of *Cl. tetani*. One was temperature and the other was temperature plus the variants that have been studied. In some instances a given temperature was sufficiently high to be effective irrespective of the variable.

THERMAL DEATH POINT. II. Spores of *Bacillus anthracis*. T. J. Murray. Jour. Inf. Dis., xlviii (1931), 5, p. 457.

Effective temperatures for the destruction of the spores of *B. anthracis* in physiologic sodium chlorid were 105° C. (5 to 10

minutes), 100° C. (5 to 10 minutes), 95° C. (10 to 25 minutes) and 90° C. (15 to 45 minutes). Effective temperatures for dry spores heated in the absence of moisture were 135° C. (5 to 10 minutes), 125° C. (25 minutes), and 120° C. (40 to 45 minutes). Dried spores heated under moist conditions showed no increased resistance. Spores of B. *anthracis* showed best resistance in all cases at pH 8. The spores of *B. anthracis* suspended in a 1 per cent peptone solution showed increased resistance as the value of the pH of the solution increased. When the effects of different concentrations of peptone were compared, the greatest resistance seemed to be shown at a concentration of 1 per cent. The resistance of spores of *B. anthracis* suspended in sodium chlorid solution seemed to decrease with increase in the concentration of the salt.

THERMAL DEATH POINT. III. Spores of *Clostridium welchii*. Mary R. Headlee. Jour. Inf. Dis., xlviii (1931), 5, p. 468.

Spores of *Cl. welchii* in physiologic sodium chlorid were killed at 90° C. (30 minutes), 95° C. (ten minutes) and 100° C. (five minutes or less). Spores were found to be more tolerant of acid than alkali. Spores in a solution of sodium chlorid at increasing concentrations of the salt to 4 per cent showed an optimum resistance at 3 per cent. At 10 per cent there was a definite decrease in resistance. Spores in peptone and gelatin solutions showed resistance inversely proportional to the concentration of organic substance at pH 5, 7 and 10. An optimum resistance was shown in 1 per cent starch. Over a period of two months spores changed little in thermal resistance when kept at ice-box temperature for the latter half of the period. Dry spores showed a resistance of 15 minutes at 130° C. and of 5 minutes or less at 140° C.

AN ANALYSIS OF THE REPUTED PATHOGENICITY OF THYSANOSOMA ACTINIOIDES IN ADULT SHEEP. Reed Christensen. Jour. Agr. Res., xlii (1931), 4, p. 245.

Postmortem studies of over 1200 sheep reported have shown that in well organized feed-lots no great damage is done to the host except where excessively heavy infections occur. In cases of heavy infection the symptoms simulate those of malnutrition and a condition described as "overeating" by Newsom and Cross. The liver appears to be part of the normal habitat of

the parasite, whether cyclic or not cannot be said, but it occurs there almost as frequently as in the intestines. The area in which *Thysanosoma actinioides* infection occurs is gradually increasing. Little is known concerning the life cycle of *T. actinioides* except that it probably requires an intermediate host.

URINARY CALCULI IN SHEEP. B. F. Pontius, R. H. Carr and L. P. Doyle. Jour. Agr. Res., xlii (1931), 7, p. 433.

Calculi were found in the kidneys, ureters, bladder and urethra, but in only a few cases were they found in all of these parts in the same sheep. The calculi varied widely in size and degree of hardness and were composed largely of calcium phosphate and organic matter. Calculi were found more often in the processus urethrae than in any other part. The S-curvature of the urethra in the male sheep offers a favorable site for lodgement of calculi. Partial or complete inability to urinate, wetness about the end of the sheath and in severe cases the arching of the back were the most frequent symptoms observed. Experiments designed to determine how different rations would affect the reaction or decomposition of urine were conducted. The reaction of fresh urine varied from pH 6.8 to 9.0 depending on the feed of the animal. Sheep on alfalfa or beet rations produced a very basic urine ash, but when a cereal grain or bran was added to the ration the reaction of the urine was nearly neutral. When the ration was utilized to the best advantage by the sheep, about three per cent of solids was found in the urine; on the more undesirable rations this increased to as high as 8 per cent of solids. In this latter instance the urine ash was very basic and it seems probable that such urine would be highly irritating to the urinary tract.

THE EFFECT OF MIXTURES OF TISSUE EXTRACTS AND BLOOD SERA OF VARIOUS ANIMALS ON THE COAGULATION OF BLOOD. E. L. Burns, F. H. Scharles and L. F. Aitken. Amer. Jour. Physiol., xcvii (1931), 2, p. 233.

Incubation of various blood sera and kidney extracts produces the following effects on coagulation of dog-heparinized plasma: (a) marked inhibition of clotting, preceded in some cases by acceleration; (b) marked acceleration of clotting. Evidence points to the development of two substances in mixtures of serum and extract, one causing inhibition and the other acceleration of coagulation. Both inhibiting and accelerating substances form

the combinations of precursor substances in blood sera and tissue extracts. Both accelerating and inhibiting precursor substances are present in all sera and extracts tested, but they vary in amount and activity in different species. The effect of the mixtures on coagulation is the resultant action of these two substances. There is evidence of a specific adaptation between both accelerating and inhibiting substances. The inhibiting precursor substances tend to disappear in the blood serum of phosphorus-poisoned dogs.

Complement Fixation with Urine in Tuberculosis. Marion E. Parker. Amer. Rev. Tuber., xxiii (1931), 6, p. 733.

Four hundred eighty-three complement-fixation tests for the detection of tuberculosis were performed on urines of tuberculous and non-tuberculous individuals. The results were compared with clinical findings in each case. The presence of specific antigens or antibodies in the urine, as demonstrated by complement fixation, is highly indicative of a tuberculous lesion somewhere in the body, but not necessarily in the urinary system. A negative test has no diagnostic significance.

Vibrionic Enteritis in Calves. F. S. Jones and Ralph B. Little. Jour. Exp. Med., liii (1931), 6, p. 845.

The authors describe an intestinal disorder of calves. The clinical manifestations are usually observed in calves two or more weeks old. Experiments indicate that infection may take place relatively early in life and may for a time produce only a mild reaction, but as the disease becomes more chronic the clinical manifestations become more pronounced. The anterior portion of the jejunum is the primary locus of infection but in more chronic cases practically the whole small intestine may be involved. Vibrios were cultivated from the inflamed intestinal mucosa in both the acute and more chronic spontaneous cases. Vibrios were obtained also from the acutely involved intestine of young calves experimentally exposed to natural infection. On three occasions similar vibrios were found in cultures from the liver. When a single strain of vibrios which had been under cultivation for three months was fed a young calf, subclinical infection was produced and the organism recovered. This strain, after three passages on media, when fed to a calf, produced a severe

inflammation of the jejunum and ileum and from these areas the organism was recovered.

VIBRIOS (*Vibrio jejunum*, n. sp.) ASSOCIATED WITH INTESTINAL DISORDERS OF COWS AND CALVES. F. S. Jones, Marion Orcutt and Ralph B. Little. Jour. Exp. Med., liii (1931), 6, p. 853.

A number of vibrios obtained from the small intestines of calves fed feces from spontaneous diarrhea in cows, natural intestinal disorders of calves, experimentally induced infections in calves and cultures obtained from Dr. Theobald Smith have been studied. From the close morphological resemblance, similarities in motility, position and number of flagella, tinctorial properties, and the tendency to fragmentation in older cultures, as well as the narrow nutritive requirements, it would seem that they belong to a very closely allied group and the authors propose the name *Vibrio jejunum*. Immunologically, as judged by agglutination, the organisms have been divided into two groups, the smaller representing two strains originating from diarrhea in cows and the larger comprising one from this source and many from the calf disease. The larger group can be subdivided by means of agglutinin absorption into cultures which do not contain the complete antigenic complex and others which do so. Certain freshly isolated vibrios, when injected into rabbits, incite a definite reaction terminating in a localization in the small intestine accompanied by catarrhal inflammation.

THE HISTOLOGY OF EXPERIMENTAL TUBERCULOSIS IN DIFFERENT SPECIES. William H. Feldman. Arch. Path., xi (1931), 6, p. 896.

With the use of pure strains of *Mycobacterium tuberculosis*, representative of the human, bovine, and avian forms of the organism, experimental tuberculosis infection was induced by various procedures in chickens, rabbits, guinea pigs and dogs. From the lesions obtained a comparative study was made to determine whether the anatomic character of the cellular reaction in the respective species varied with the type of tubercle bacillus used to incite the lesions or whether the histologic character of the lesions in a given species was constant regardless of the form of tubercle bacillus present. Variations in the character of the reaction of the tissue to *Mycobacterium tuber-*

culosis are not determined by the type of the organism *per se*, but by certain indefinite factors which are inherent in the species possessing the infection. The histologic nature of the tuberculous infection in a given species is essentially the same regardless of the form or of the origin of the particular strain of tubercle bacillus responsible for the lesions. The lesions of tuberculosis in the common fowl usually possess certain anatomic characters that distinguish them from tuberculous lesions in the lower animals. The author concludes that his observations indicate that it would be injudicious to designate a given tuberculous infection as being due to organisms of human, avian, or bovine origin, in the absence of results of tests for pathogenicity.

EDEMA. Herman Elwyn. Abst. in Arch. Path., xi (1931), 6, p. 949.

There is a general understanding that in every case of general edema, there is a slowing up in the rate of movement of water from depots through the tissue barriers to the blood-stream. With the normal intake of water and salt and their rapid removal from the blood into the tissues, the free water there is rapidly increased. When a certain amount of water has accumulated it becomes visible as edema. The slowing is the result of a central regulatory change and affects the constellation of electrolytes in the tissue barriers through which the water moves to the blood- and lymph-streams. This slowing is produced, first, whenever the water content of the blood threatens to be unduly increased, as in cardiac failure, or in acute diffuse nephritis; second, when the change is primarily in the regulatory center, along with the depression of other vegetative functions especially of the basal metabolic rate. This is the case in chronic undernutrition and in chronic wasting disease. Third, it is produced when there is a primary disturbance in the central regulation for the control of sodium chlorid in the body. This is so in certain cases of edema without cardiac or renal diseases, and without chronic undernutrition.

ANTERIOR PITUITARY THERAPY IN SPONTANEOUS OVARIAN DYSFUNCTION. Warren O. Nelson. Endocrinol., xv (1931), 2, p. 128.

Eight adult female rats, which for at least five weeks had shown no estrous changes in their vaginal smears, were given implants of anterior lobe or daily injections of an extract prepared from

anterior lobe material. Estrum occurred in each instance on the second and third days subsequent to the instigation of the treatment. The ovaries and uteri prior to treatment presented a picture approximating that of diestrum, while the remaining ovaries and uteri, removed subsequent to the occurrence of the induced estrum, were either of an estrous or meta-estrous character. When copulation was permitted at the time of induced estrum, successful pregnancies resulted. The pituitaries of these females were capable of promoting precocious maturity in a young rat when introduced intramuscularly. No further estrous changes could be demonstrated beyond the initial induced estrum. It is concluded that the etiology of the aberrant ovarian behavior observed in these animals probably was some derangement of the normal hypophysial-gonadal relationship, possibly the non-release of the gonadal stimulating hormone. The author points out the possible therapeutic value of anterior lobe treatment in some types of sterility.

THE FEMALE HORMONE (MENFORMONE) IN MALE ORGANS; SOME VIEWS ON SPECIFICITY. S. E. deJongh, E. Laqueur and E. Dingemanse. Abst. in Endocrinol., xv (1931), 2, p. 159.

Menformone was readily obtained from male urine by benzene extraction, saponification and solution of the product in water. The potency was one mouse unit per 0.02 mgm. This material was injected into a variety of animals. It caused a marked increase in the size of the female genitals and mammary glands, had an antimasculine influence, stimulated vital activities of females and was entirely non-toxic. The apparent differences in effect on males and females are probably due to the higher concentration in the latter. Two phases of specificity are emphasized, one based upon the characteristic properties and the other on origin.

THE MORPHOLOGIC CHANGES IN ANIMAL TISSUES DUE TO HEATING BY THE ULTRA HIGH-FREQUENCY OSCILLATOR. Victor C. Jacobsen and Keijoshi Hosoi. Arch. Path., xi (1931), 5, p. 744.

A study was made of the effects on animal tissues of exposure in the electrostatic field of a high-frequency oscillator. Twenty-three dogs, twenty-seven white rats and three guinea pigs were used. A marked hyperthermia was produced in the animals,

the amount of temperature increase being controllable by altering either the voltage or the distance between the plate electrodes of the apparatus. The morphologic changes occurring in the tissues were congestion of the organs, peripheral hyperemia, cloudy swelling, fatty degeneration, dehydration, glycogen depletion, focal hemorrhage especially in the gastro-intestinal tract, epithelial hyperplasia in the parenchymatous organs and stimulation of the bone marrow. Following prolonged periods of heating, degenerative lesions occurred in the male germinal epithelium. The alterations observed differ but little from the effects of fever produced by various other methods, but the ease with which the temperature can be controlled in the high-frequency field of this apparatus and the character of some of the tissue responses suggest possible therapeutic application.

VACCINATION OF MAN AGAINST UNDULANT FEVER. C. Dubois and M. Sollier. Abst. in Arch. Path., xi (1931), 5, p. 815.

Preventive vaccination has been accomplished on 111 persons who had been exposed to Brucella infection (caprine, ovine or bovine). No subjects vaccinated from 3 to 8 months previously have actually contracted undulant fever. Two of 36 non-vaccinated controls were attacked. It is thus concluded that in regions where animal brucelliasis exists, preventive vaccination is the only practical means of protecting man. In "contaminated" regions vaccination should be practiced on those coming in contact with infected animals. The vaccines consisted of heat-killed suspensions of human, ovine, caprine, porcine and bovine cultures at a total of two billion organisms per cubic centimeter. The duration of immunity can be determined only by longer experience with more vaccinations.

PHYSIOLOGIC AND BIOCHEMICAL CHANGES RESULTING FROM EXPOSURE TO AN ULTRA HIGH-FREQUENCY FIELD. Arthur Knudson and Philip J. Schaible. Arch. Path., xi (1931), 5, p. 728.

The body temperature of dogs can be raised to any desired point by exposure to an ultra high-frequency field. The temperature returns to normal rapidly unless the animal has been heated above 42° C. Animals heated above 42.5° C. for any great length of time do not survive. Exposure of animals to an ultra high-frequency field results in loss of weight and decrease

in blood volume which usually returned to normal within 24 hours. Raising the temperature of animals to 41.7° C. does not produce any great change in hydrogen-ion content of the plasma, although there is a tendency toward a condition of alkalosis. With higher temperatures the tendency is toward a condition of acidosis, probably caused by increase in the production of lactic acid. Changes in chlorids, total protein and total base are accounted for by the dehydration. Bicarbonate was greatly reduced. Changes in inorganic phosphorus were somewhat variable. Blood sugar, non-protein nitrogen in the blood, urea nitrogen, creatinine and amino-acid nitrogen are also increased. There is an increase in both red blood cells and total white cells. Often there is an increase in immature forms of red cells.

U. S. Civil Service Examination

The United States Civil Service Commission announces an open competitive examination for assistant veterinarian (bacteriology) and junior veterinarian (bacteriology) to fill vacancies in the Bureau of Animal Industry, Department of Agriculture, for duty in Washington, D. C., or in the field. The entrance salary for assistant veterinarian ranges from $2,600 to $3,200 a year, and for junior veterinarian it ranges from $2,000 to $2,600 a year.

Competitors in the assistant examination will be rated on answers to practical questions in veterinary bacteriology, and on their education, training and experience; in the junior examination competitors will be rated on the same subjects, with the exception of experience. Graduation from a veterinary college of recognized standing is required. Two years of certain post-graduate study or suitable experience is also required of applicants for the assistant examination.

Applications for examination must be on file with the Civil Service Commission at Washington, D. C., not later than October 1, 1931.

Full information may be obtained from the Secretary of the United States Civil Service Board of Examiners at the post office or customhouse in any city or from the United States Civil Service Commission, Washington, D. C.

Regular Army

Captain Peter T. Carpenter is relieved from further assignment and duty at the veterinary general dispensary, Washington, D. C., and additional duty as attending veterinarian at the Army War College, Washington, D. C., Bolling Field, D. C., Fort Washington, Md., Fort Hunt, Va., and Fort Humphreys, Va., and is assigned to Fort Sheridan, Ill., for duty, upon the expiration of the leave of absence heretofore granted him.

Captain Frank B. Steinkolk is relieved from further assignment and duty at Fort Sheridan, Ill., effective on or about October 15, 1931, will proceed to Chicago, Ill., and report to the commanding officer Chicago general depot for duty, and in addition will report to the commanding general Sixth Corps Area for duty at his headquarters.

Captain John K. McConeghy is relieved from duty at Fort Robinson, Nebr., effective at such time as will enable him to sail from San Francisco, Calif., on transport scheduled to leave that port on or about November 25, 1931, for the Philippine Department.

The appointment and assignment of Second Lieutenant George Townley Price, Veterinary Corps Reserve, as second lieutenant in the Veterinary Corps, Regular Army, with rank from July 21, 1931, is announced. He will proceed from New Cumberland, Pa., to Carlisle Barracks, Pa., and report to the commanding officer that station for duty.

The promotion of the following-named officers to the grade of Major is announced:

Captain Gerald W. FitzGerald
Captain Stanley C. Smock
Captain Charles B. Dunphy
Captain Howard N. Beeman
Captain Howard M. Savage
Captain Burlin C. Bridges
Captain Mott Ramsey
Captain Josiah W. Worthington
Captain Fred C. Waters
Captain Harry E. Van Tuyl

The promotion of 2nd Lieutenant Maurice W. Hale, to the grade of 1st lieutenant with rank from July 25, 1931, is announced.

Veterinary Reserve Corps

Promotions

Barrett, Clinton DeLos....To Capt...490 E. Cuyahoga Falls Ave., Akron. O.
Exconde, Irineo Imperial....1st Lt...San Pablo, Laguna, P. I.
Reinhardt, Wade Hampton..Major...114 So. Myers St., Charlotte, N. C.

New Acceptances

Ash, Harley Edwin.....1st Lt...204 So. Summit St., Bowling Green, O.
Aitken, Howard M.....2nd Lt...127 Wilson Road, Camp Chase, O.

Bailey, Leonard Perry	2nd Lt.	R. D. No. 2, South Charleston, O.
Benner, Kenneth Leland	2nd Lt.	R. R. No. 3, Bainbridge, O.
Bennett, Paul Clifford	2nd Lt.	212 Grand St., Morgantown, W. Va.
Bowen, William Clinton	2nd Lt.	County Hospital, Urbana, O.
Burris, James Thomas	2nd Lt.	Smithfield, Ohio
Burriss, Kenneth Kent	2nd Lt.	Smithfield, Ohio
Cellar, Paul Markel	2nd Lt.	R. F. D. No. 6, Ashland, Ohio
Fenner, Walter Harmon	2nd Lt.	821 West Main St., Hillsboro, O.
Ferrall, Walter Candle	2nd Lt.	33 Duquesne St., Columbiana, O.
Gluhm, Charles Christian	2nd Lt.	625 Prytania Ave., Hamilton, O.
Hancock, Amor Edward	2nd Lt.	2147 Neil Ave., Columbus, O.
Pastors, Charles Robert	2nd Lt.	Rayland, O.
Peters, James Ralph	2nd Lt.	R. R. No. 7, Fremont, O.
Putnam, John Lyle	2nd Lt.	Frankfort, Ohio
Richardson, Leonard Robert	2nd Lt.	R. F. D. No. 8, Box 35, Akron, O.
Leasure, Thomas Joy	2nd Lt.	837 New Hampshire St., Lawrence, Ks.
Armstrong, Robert Paris	2nd Lt.	105 S. Earl St., Shippensburg, Pa.
Murphy, John Joseph	2nd Lt.	488 Pleasant St., Malden, Mass.
Witt, Raymond Francis	2nd Lt.	1253 Main St., Worcester, Mass.

Convention Notes

Dr. George B. Vliet, of Hackettstown, N. J., was the only member in attendance from the Garden State.

The New York State Veterinary College at Cornell University was represented by Drs. R. R. Birch, E. L. Brunett, J. W. Benner and W. A. Hagan. Dr. Birch represented Dr. D. H. Udall at the Executive Board meetings.

Dr. Robert S. MacKellar, of New York City, was reelected chairman of the Executive Board for the ensuing year. He concluded his fourth year as member, and his first year as chairman of the Board, at Kansas City.

Amoung the state veterinarians seen at the meeting were: Cary, of Alabama; Bux, of Arkansas; White, of Idaho; Brown, of Indiana; Malcolm, of Iowa; Cotton, of Minnesota; Bradshaw, of Mississippi; Wilson, of Missouri; Crewe, of North Dakota; Lytle, of Oregon; Munce, of Pennsylvania; Williams, of Texas; and Wisnicky, of Wisconsin.

New Dean at Pennsylvania

The School of Veterinary Medicine at the University of Pennsylvania has a new dean in the person of Dr. George Alexander Dick (U. P. '04), recently appointed to fill the vacancy caused by the death of Dr. H. E. Bemis earlier in the year. In the October issue of the JOURNAL we will have more to say about Dean Dick.

MARYLAND STATE VETERINARY MEDICAL ASSOCIATION

The sixteenth semi-annual meeting of the Maryland State Veterinary Medical Association was held at the University of Maryland, College Park, June 24-25, 1931, with about 75 members and guests in attendance.

The presidential address was delivered by Dr. Hulbert Young, of Baltimore. After extending a hearty welcome to the members and friends, the President congratulated the ladies on the first anniversary of their auxiliary. The theme of the address was skillfully developed around two thoughts which were (1) our responsibility to the profession and (2) a plea for closer contact with and greater efforts in research.

The second paper was entitled, "The Effect of Pasteurizing Temperatures on (a) *Brucella Abortus* and (b) *Brucella Abortus* Agglutinins in Milk," by Mr. Thomas Bartram, Department of Bacteriology, University of Maryland. As Mr. Bartram was away on vacation, the paper was read by Dr. R. C. Reed. This paper will appear at an early date in the *Cornell Veterinarian* and is, therefore, not reported in detail here.

The third paper was entitled, "Rabies Immunization," and was delivered by Major R. A. Kelser, Veterinary Corps, U. S. Army. It was abstracted by the author as follows:

> While vaccination of dogs is of definite value in the control of rabies, and is to be commended, its application should not be responsible for a let-up in general control measures of established merit.
>
> Single doses of properly prepared vaccine will immunize many dogs against rabies but it should not be assumed that single-dose vaccination will invariably protect every animal against any and all types of natural infection. It is pointed out that it is impossible definitely to compare artificial infection by hypodermic injection of nerve-tissue containing rabies virus, with natural infection when virulent saliva is introduced into tissues injured in various ways and degrees by laceration, contusion, secondary infection, etc.
>
> For general vaccination on a wide-spread scale the single-dose method, obviously, has much to recommend it from the standpoint of practical application. Experimental data and such evidence as is available from

field use of the vaccine indicate the value of single-dose vaccination. However, the administration of two or three doses of vaccine, with intervals of 5 to 7 days between doses, will confer greater immunity and is recommended for individual cases in which the additional injections present no problem of practicability. This latter practice is especially desirable in localities where rabies is particularly prevalent.

Within certain limits, rabies vaccine used in the single-dose method of immunization varies in efficacy in proportion to its nerve-tissue content. Chloroform-treated vaccine containing 33⅓ per cent nerve tissue has proved superior to phenol-treated vaccines.

The speaker doubts the existence of different types of street virus and attributes apparent differences simply to marked variation in virulence.

The fourth paper was entitled, "Notes on the American Veterinary Medical Association European Tour—1930." It was presented by Dr. W. E. Cotton, U. S. B. A. I. Experiment Station, Bethesda. The author gave a graphic account of the meeting and the extra-curricular activities (social functions) as well as of the tour following the meeting. It was most interesting and instructive and the reporter is inclined to believe that the usually somewhat serious-minded author at least observed some of the lighter functions rather closely.

The afternoon was devoted to clinics. Dr. I. M. Cashell, Washington, D. C., conducted the small-animal work. Dr. W. F. Guard, Ohio State University, had charge of the large-animal part. Both branches were most interesting and instructive.

The banquet was held in the evening at the University Dining Hall. Dr. Hulbert Young acted as toastmaster. The speakers were Hon. S. M. Shoemaker, Chairman, Board of Regents, University of Maryland; Dr. R. A. Pearson, President, University of Maryland; Dr. W. F. Guard, Ohio State University; Prof. Charles S. Richardson, Professor of Public Speaking, University of Maryland; Dr. J. E. Shillinger, Bureau of Biological Survey, U. S. Department of Agriculture, and others. There were about fifty in attendance.

The first paper on the second day was given by Dr. E. B. Simonds, U. S. Inspector in Charge Tuberculosis Eradication, Baltimore, and was entitled, "Testing Accredited Herds." It was abstracted by the author as follows:

After a herd is accredited, the contact between the accredited veterinarian and the owner is much closer than is our association with him and he looks to you for advice as to the handling of his cattle and general conduct of his herd. As the success of tuberculosis eradication depends to a great extent upon keeping these herds clean, and as you accredited veterinarians have a vital financial, as well as public interest in this work, we as well as the owners of the accredited herds are entirely dependent upon you as to the proper conduct of the work.

We believe that about 75 per cent of the accredited herds in which reactors are found are infected by introduction of new cattle. In some instances these herd additions have never been tested. Others have been

tested but come from badly infected herds and still others come from clean herds but have been in dealers' infected stables long enough to pick up the disease and carry it into the accredited herds. Each owner should be warned of the trouble that careless buying may cause him, and should be instructed that when he buys cattle they must come directly from accredited herds, and if they do not, that they must come from clean herds, tested when he buys them, and held in quarantine for sixty days and retested before being placed in his herd.

The second paper was delivered by Dr. F. W. Tilley, Biochemic Division, U. S. Department of Agriculture. It was entitled, "Disinfection." Dr. Tilley emphasized the fact that disinfectants are not general but specific in their action. As examples he cited differences in the efficiency of phenol and of sodium hydroxid against certain bacteria on the one hand and against viruses of hog cholera and foot-and-mouth disease on the other. He also called attention to the effect of organic matter and other modifying factors and showed that disinfectants do not act instantaneously but require an appreciable length of time for efficient action. Attention was also called to the importance of cleaning up before disinfecting. In closing he stated that disinfection must not be expected to do everything in preventing the spread of disease but is useful in coördination with other measures.

The last paper on the morning program was delivered by Dr. W. F. Guard, and was entitled, "Veterinary Surgery." The subjects treated were as follows:

First, the roaring operation was discussed and attention called to the mistake that often is made in operating on animals that are not true roarers. The objects of the operation and the necessity for carefully carrying out in detail the proper technic were stressed. The anatomy and physiology of the larynx were briefly discussed. It was also pointed out that in the author's opinion the operation can not be performed as carefully, nor as thoroughly carried out in the standing position as compared to the recumbent position under an anesthetic.

Second, flattened or collapsed trachea. A case was reported in which a trachea was collapsed for about twelve inches just anterior to the entrance to the thorax. The animal was operated in a standing position with the use of a local anesthetic. An incision about twelve inches long was made on either side of the neck, exposing the two edges of the trachea. The skin and muscles were retracted near the center of the area so as to expose the anterior surface of the trachea. The interannular ligament was partially severed between two cartilages at about the middle

of the collapsed area, in order to permit passing a finger inside the trachea to direct the succeeding steps of the operation.

A long straight sac needle, threaded with silver wire suture material, was then passed through the lumen of the trachea at the lowest point of the collapsed portion. The needle was then returned through the lumen of the trachea and passed around two cartilaginous rings. Four such sutures were placed through the lumen of the trachea so as to cover the entire collapsed portion. The anterior surface of the cartilaginous rings was then slightly nicked with a scalpel in order to weaken the anterior wall. The wire staple sutures were then pulled together so as to cause the trachea to take on its proper shape. These were twisted and the ends permitted to stick out through the edges of the wound. The wound was then sutured and a protective dressing applied over the region. The wire sutures were left in position seven days. At this time they were removed. The wounds were completely healed in about four weeks and the animal apparently made a complete recovery.

Third, the subject of choke in the horse and ox was discussed. For those cases of low choke, the speaker recommended the passing of a stomach-tube, making an incision through the skin and over-lying structures about the middle of the neck until the esophagus was exposed and isolated. Then pass a piece of two-inch bandage around the esophagus and ligate with the tube in position. Then, by the use of the stomach-pump and a gallon of mineral oil, proceed cautiously to dilate and lubricate the mass and force it into the stomach.

In those cases of choke in the cervical portion of the esophagus, the speaker recommended the use of Williams' horse trocar, and described the technic of breaking up the choke mass, and in case of cows choking on ears of corn, the possibility of correcting the position of the object and pushing it back to the pharynx where it could be removed.

The subject of the use of the stomach-tube in cattle was also discussed and demonstrated. The tube recommended was a heavy steam hose with one-inch inside diameter which was to be carried in a coil. The mouth-gag was merely a block of wood shaped to fit the mouth of the cow, with a hole in the center.

Fourth, the value of the colorimetric test and physical examination of the udder, in order to detect and control mastitis, was discussed. The use of brom-thymol-blue indicator, as recommended by Dr. D. H. Udall, is used in the test. One gram of

brom-thymol-blue, added to 500 cc of 47½% alcohol, constituted the indicator. However, this indicator must be near the neutral point. It may be necessary to add a small amount of normal sodium hydroxid solution in order to bring the solution to the proper reaction.

The second afternoon was devoted to large-animal clinics. Dr. Guard was again in charge. The members of the Association were so interested in the operations that the meeting was not adjourned until 5:30 o'clock.

E. M. PICKENS, *Secretary-Treasurer.*

KENTUCKY VETERINARY MEDICAL ASSOCIATION

The summer meeting of the Kentucky Veterinary Medical Association was held at the Seelbach Hotel, Louisville, July 8-9, 1931. About 80 members and a number of guests attended the sessions. The Committee on Program was highly complimented for the interesting and valuable program which had been arranged. Dr. F. H. Riester, of Buechel, presided throughout the meeting.

The first speaker was Dr. C. H. Harris, Director of Public Health, Louisville, who represented Hon. William Harrison, Mayor, for the address of welcome. In his talk, Dr. Harris gave an outline of the improved milk ordinance, now in effect in Louisville. Dr. Harris stressed the important part the veterinarian plays in this work, and he expressed a strong desire to coöperate with veterinarians, in an effort to give Louisville a wholesome milk supply.

The response to the address of welcome was made by Dr. T. P. Polk, of Lexington, who placed great emphasis on the importance of milk inspection, starting with a thorough physical examination of the cow, made by a qualified veterinarian.

The principal speaker of the meeting was Dr. R. R. Dykstra, of Kansas State College, who spoke on general veterinary observations, during the afternoon session of the first day, and discussed some equine problems, during the morning session of the second day. A rising vote of thanks was extended Dr. Dykstra by the Association, for his interesting and instructive talks. Those present were profuse in their praise of the clear, concise way in which Dr. Dykstra presented his subjects, and the amount of ground that he was able to cover in the time allotted.

Dr. J. A. Winkler, of Newport, presented a very valuable paper on canine practice and hospitalization.

The Association voted in favor of the proposed plan for affiliation with the American Veterinary Medical Association.

The election of officers for the ensuing year resulted in the unanimous choice of the following: President, Dr. A. J. Steiner, Lexington; first vice-president, Dr. R. H. Bardwell, Lexington; second vice-president, Dr. E. A. Caslick, Paris; third vice-president, Dr. C. H. Mainhart, Richmond; secretary-treasurer, Dr. J. R. Stifler, Lebanon.

The evening of the first day was devoted to a conference of co-operating state and federal veterinarians, to which all practicing veterinarians were invited. Tuberculosis eradication, sheep scab control and the hog cholera situation were among the matters discussed at the conference, which was concluded with a question-box.

J. R. STIFLER, *Secretary-Treasurer.*

VIRGINIA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-eighth annual meeting of the Virginia State Veterinary Medical Association was held at Richmond, July 9-10, 1931. From the standpoint of attendance, this meeting was probably the most successful ever held.

It was voted to adopt the plan submitted by the A. V. M. A. Special Committee on Affiliation, and a member was elected to the House of Representatives.

It was voted to accept the invitation of the North Carolina State Veterinary Medical Association for a joint meeting at Henderson, N. C., June 29-30, 1932.

The following papers were presented:

"The Use of Calcium Chlorid in Veterinary Practice," by Dr. Herbert Howard, Leesburg, Va.

"Canine Distemper: Its Prevention and Treatment," by Dr. Norman J. Pyle, Lederle Laboratories, Inc., Pearl River, N. Y.

"Some Modern Methods," Dr. Ashe Lockhart, Kansas City, Mo.

"A New Method of Vaccinating Against Chicken-Pox," by Dr. E. P. Johnson, Blacksburg, Va.

A banquet was held at Murphy's Hotel, the evening of the first day, Dr. J. P. McDonough, of Richmond, acting as toastmaster. Dean H. L. Price, School of Agriculture, Virginia Polytechnic Institute, Blacksburg, delivered an address, "The Fifth Estate and the Veterinary Profession." Dr. J. E. Shillinger of the U. S.

Bureau of Biological Survey, Washington, D. C., presented a motion-picture film, entitled, "The Fur Industry of the United States."

On the second day of the meeting a clinic was held at the veterinary hospital of Dr. McDonough, under the direction of Dr. J. P. Hutton, of Michigan State College. At noon, luncheon was served at the home of Dr. H. H. Rowe, by himself and his brother, Dr. Taylor P. Rowe.

Officers for the ensuing year were elected as follows: President, Dr. W. H. Grubb, Purcellville; first vice-president, Dr. P. M. Graves, Culpepper; second vice-president, Dr. Herbert Howard, Leesburg; secretary, Dr. I. D. Wilson, Blacksburg; treasurer, Dr. R. E. Brookbank, Richmond.

I. D. Wilson, *Secretary.*

NORTHWESTERN PENNSYLVANIA VETERINARY CLUB

The Northwestern Pennsylvania Veterinary Club met at the home of Dr. E. E. Bittles, Waterford, Pa., July 14, 1931.

Dr. Nevin S. Craver, of Youngstown, Ohio, gave a very instructive talk on small-animal practice. Dr. R. M. Phalen, of Sharon, Pa., discussed cases of interest in connection with cattle practice. These discussions brought out many practical points and valuable information, helpful to those present.

Dr. T. E. Munce, of Harrisburg, spoke on the work of the Pennsylvania Bureau of Animal Industry, with special reference to new legislation. Dr. John Bryce, of Erie, Pa., a graduate of the Ontario Veterinary College, class of 1870, who is believed to be one of the oldest living graduate veterinarians, having attained the age of 86 years, took an active part in the proceedings and, regardless of his physical disability, he continues to take a deep interest in veterinary matters, as well as local affairs.

About 28 veterinarians from northwestern Pennsylvania, northwestern Ohio and southwestern New York were in attendance.

B. Scott Fritz, *Reporter.*

THE VETERINARY ASSOCIATION OF MANITOBA

The annual summer clinic of the Veterinary Association of Manitoba was held at Brandon, in the Winter Fair Building, July 25, 1931. An attractive program was provided under the

direction of Dr. H. H. Ross, of Brandon. There was a good attendance of practitioners, from all parts of the Province.

The proceedings were opened with an address of welcome from Mayor Cater, of Brandon. The chief operator and demonstrator was Dr. T. H. Ferguson, of Lake Geneva, Wis., who performed the following operations: Rumenotomy, umbilical hernia in a filly, actinomycosis in a bull, dental cyst in a horse, diagnosis of lameness and so forth. Dr. Ferguson was assisted by Drs. H. R. McEwen, of Stonewall; J. W. Fasken, Portage la Prairie; W. F. Sirett, Minnedosa; S. J. Gibson, Brandon; and H. Bradshaw, Portage la Prairie. Dr. Gibson also gave a very able demonstration of methods of restraint.

In the evening a banquet was held at the Prince Edward Hotel. Dr. W. A. Shoults occupied the chair, and addresses were given by Drs. T. H. Ferguson, H. R. McEwen and M. T. Lewis. These were interspersed with musical numbers which were appreciated by all present.

A hearty vote of thanks was given Dr. Ferguson, for his contributions to the program, and a resolution of regret was adopted on the inability of the President, Dr. J. B. Still, to be at the clinic, his absence being on account of ill health.

WM. HILTON, *Secretary-Treasurer.*

BAY COUNTIES VETERINARY MEDICAL ASSOCIATION

The Bay Counties Veterinary Medical Association held a special dinner meeting, Monday evening, August 3, 1931, at the Elks Club, San Francisco, Calif., in honor of Dr. John R. Mohler, chief of the United States Bureau of Animal Industry, who had been spending a vacation in California.

Besides many members and a number of visitors, the meeting was attended by representatives of the various branches of the Bureau of Animal Industry in the state of California. Dr. Mohler was the principal speaker of the evening, his topic being "Recent Advances in the Study of Infectious Abortion in Cattle."

GEO. M. SIMMONS, *Secretary.*

MISSISSIPPI VETERINARY MEDICAL ASSOCIATION

The annual picnic of the Mississippi Valley Veterinary Medical Association was held at Monmouth, Ill., August 5, 1931. Veterinarians, their wives and members of their families were in attendance to the number of approximately 150.

A big feature of the day's entertainment was a musical concert given by Kirkwood High School orchestra, of fifteen pieces, under the leadership of Mr. Bruce, of Burlington, Iowa. No program was attempted, as the meeting was a picnic, pure and simple.

EAST TENNESSEE VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the East Tennessee Veterinary Medical Society was held at Knoxville, August 8, 1931.

Dr. R. G. Kern, local surgeon, made the principal address of the evening, on "Newer Things in Human Surgery," bringing out the most important recent developments in the field of human surgery and commenting on how these might be applied to veterinary surgery.

Dr. D. Coughlin discussed "The Intradermic Tuberculin Test," and Dr. G. A. Metcalf spoke on "The Practical Application of the Agglutination Test in the Control of Bang's Disease."

NEVADA STATE VETERINARY ASSOCIATION

The mid-year meeting of the Nevada State Veterinary Association was held at the University of Nevada, Reno, August 10, 1931. Dr. Maurice C. Hall, president of the American Veterinary Medical Association, was the guest of honor.

Following a business session, Dr. Hall presented to the Association his tentative program of action for the A. V. M. A., which he hoped would be translated in terms of individual and cooperative efforts toward its realization.

Various members of the Association then presented a number of interesting case reports.

Dr. R. A. Given, U. S. Bureau of Animal Industry, Reno, presented "Eradication of Sarcoptic Scabies." Dr. L. R. Vawter, University of Nevada, Reno, presented "A Study of Actinomycosis." Dr. Dwight L. Hood, of Reno, gave "A General Review of the Actinomyces Group in Humans."

The Association unanimously endorsed the proposals submitted by the A. V. M. A. Special Committee on Affiliation.

WARREN B. EARL, *Secretary.*

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Medical Society was held on August 12, 1931, at Kenosha Lake, overlooking the Ashokan Reservoir. Owing to the bad weather

the attendance was materially reduced, but some thirty-five were present.

A very fine luncheon was prepared under the direction of Sheriff (Dr.) and Mrs. Smith, after which Dr. J. G. Hardenbergh, of the Walker-Gordon Laboratory Co., Inc., Plainsboro, N. J., was introduced by President Eichhorn. Dr. Hardenbergh discussed "Some Phases of Milk Control."

The speaker called attention to three diseases that have, in his opinion, the most influence on the milk business, viz., bovine tuberculosis, Bang abortion disease and mastitis.

In connection with the control of tuberculosis in dairy herds the speaker referred to the fact that some two thousand autopsies made on cattle at the Walker-Gordon Farms, destroyed after their milking efficiency was over, had revealed but eight animals that showed lesions in any way resembling tuberculosis. He stated that all cattle bought for these herds were blood-tested and that a comparatively small percentage reacted on retest. The rapid agglutination test method has been found quite satisfactory. The raising of replacements had proven very satisfactory, but the speaker was doubtful if such a plan would be practical in the average dairy.

He considers mastitis very serious and believes that early detection of the infected animal is very important. Through the use of a strip-cup, cases at Walker-Gordon Farms are often detected promptly, the animal immediately removed from the milking-line, grain ration reduced and frequent milking practiced. Various treatments have been tried with some success. The brom-thymol-blue test is considered valuable, but may be misleading under certain conditions.

The speaker referred to septic sore throat as a dangerous disease that might be transmitted through milk, and is of the opinion that cows are infected from human sources, the udder of the animal being merely a reservoir for the organism.

After some further discussion, a brief business session was held by the Society, during which time the sum of $200 was voted to be contributed by the Society for the use of the committee in connection with the International Veterinary Congress in 1934.

J. G. WILLS, *Secretary-Treasurer.*

Convention Notes

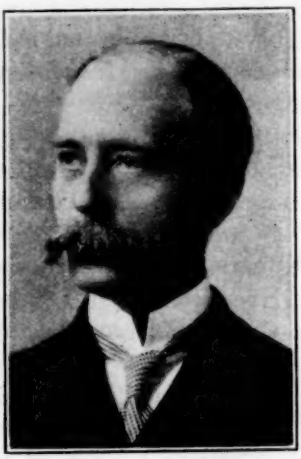
Notes on the Kansas City convention will be found on pages 331, 348, 375, 396 and 407.

NECROLOGY

ARTHUR M. FARRINGTON

The ranks of the veterinary profession again sustain the loss by death of one of its pioneers, Dr. Arthur M. Farrington, who died at his home, 1436 Chapin Street, N. W., Washington, D. C., August 3, 1931.

Dr. Farrington was born September 22, 1856, at Brewer, Penobscot County, Maine. After completing his common school education, he entered the Maine State College (now the University of Maine) and was graduated from that institution in



DR. A. M. FARRINGTON

1876 with the degree B. S. in Agriculture. He took a postgraduate course in chemistry at the same college. He then matriculated in veterinary science at Cornell University and was graduated in 1879, receiving the degree D. V. S. Thus, in the death of Dr. Farrington, Cornell again loses one of its distinguished graduates.

From 1879 to 1880, Dr. Farrington held the position of veterinary inspector on the staff of Gen. M. R. Patrick, stamping out

contagious pleuro-pneumonia in cattle in New York State. Following his employment on pleuro-pneumonia work, he became professor of agriculture at the Delaware Agricultural College. From September, 1881, to January, 1882, he held the post of veterinary inspector for the U. S. Treasury Cattle Commission.

For a short time Dr. Farrington engaged in the practice of veterinary medicine in Maine, later going to Detroit, Michigan. He again was called in government service and from 1883 to 1887 he was U. S. inspector for the port of New York and superintendent of the cattle quarantine station at Garfield, N. J. He was transferred to Washington, D. C., October 8, 1887, and made assistant chief of the Bureau of Animal Industry, remaining in that assignment until 1893. From 1893 to 1896 he was chief of the Division of Field Investigation and Miscellaneous Work; from 1896 to 1899, chief of the Miscellaneous Division; from 1899 to 1906, chief of the Inspection Division. Again he was made assistant chief of the Bureau, serving in this capacity from 1906 to 1914, when he resumed the position of chief of the Miscellaneous Division, which he held until the date of his retirement, April 19, 1921.

His connection with the Bureau of Animal Industry early in its history made Dr. Farrington one of the outstanding figures in the development of policies in that organization. His time was wholly occupied with administrative duties. His keen sense of perception, his devotion to the work at hand and his anxiety for the building up of morale made his services of untold value. For a number of years, at stated intervals, he visited the meat-inspection stations of the Bureau, holding examinations for promotion within the ranks of lay inspectors, positions termed at that time taggers and stock examiners.

In 1908, while assistant chief of the Bureau, he was made a member of the committee appointed by the Secretary of Agriculture to prescribe a standard of veterinary education. As secretary of that committee he visited all the veterinary colleges of the United States. He later was made a member of a committee within the Bureau to study the methods of teaching in the various veterinary schools of the country, and to make recommendations looking to the improvement of the standard of courses of studies.

Dr. Farrington joined the A. V. M. A. in 1892, and from 1897 until 1915 he held numerous committee assignments and appointments. From 1897 to 1900 and from 1901 to 1903 he was resident secretary for the District of Columbia. He was a member of the

Committee on Program (1904-05), member of the Committee on Publication (1905-07), member of Committee on Association of Faculties (1909-10), chairman of the Committee on Resolutions (1910-11) and chairman of the Committee on Agricultural College Investigation (1912-15).

In 1885, Dr. Farrington married Miss Addie Powers. From this union three daughters were born, Helen and Gladys, of Washington, and Ethel, now Mrs. Marsh, of New Jersey. Mrs. Farrington died July 8 of this year. Besides the three daughters, there are four brothers surviving, Dr. Oliver Farrington, of Chicago, Ill.; Edward H. Farrington, of Madison, Wis.; Horace P. Farrington, of Brooklyn, N. Y.; and Wallace R. Farrington, of Honolulu, former Governor of the Hawaiian Islands.

Doctor Farrington was a communicant of the Congregational Church and was an active member of this congregation for many years. His home life was exemplary in the extreme. An atmosphere of hospitality prevailed in the home and friends were always welcome.

T. P. W.

SHIBAMIRO KITASATO

When Professor Shibamiro Kitasato passed away at his home in Japan, last June, the world lost another of the pioneer bacteriologists who laid down the foundation of modern medicine during the last two decades of the nineteenth century.

Contemporary of Pasteur, Koch, von Behring, and let us not forget Salmon and Theobald Smith in this country, Kitasato, working in the laboratories on Continental Europe, where he was sent for study by his government in the late '80s, made his presence felt by discovering the specific organism of bubonic plauge, *Bacterium pestis*, and along with von Behring did much of the brilliant work which led up to the discovery of antitoxins.

It was Kitasato who isolated the *Clostridium tetani* (1890), six years after Nicolaier had demonstrated its presence associated with the ambient organisms of garden earth, and it was he who first attempted to attenuate bacterial toxins with chemicals. He employed trichlorid of iodin with tetanus toxin and was able to immunize 40 per cent of test rabbits, but this research was short lived as the hyperimmunization of horses to produce the product the world has enjoyed for forty years was soon forthcoming.

Kitasato is no stranger to the readers of veterinary literature of the nineteenth century, and his passing away takes one back to days when medicine, blocked by the usual army of skeptics, was turning a sharp corner trying to prove the germ theory of disease.

We younger veterinarians of the early '90s having received but a meager trace of bacteriology at college, were fascinated by the work of these authors. Slowly as the reports of their dramatic research came to us in the scant veterinary literature of that day, we literally devoured it, sensing in the new pathology not only an explanation of the baffling problems of our daily practice but also the dawn of a new era of medicine.

I am sure all of the older veterinarians will join in venerating Kitasato whose life was full of devotion to his chosen profession and whose return to his native country brought Japan among the foremost nations in medical research.

L. A. M.

PERSONALS

MARRIAGE

Dr. O. Emmitt (K. C. V. C. '06), of Wichita, Kans., to Miss Anna Thompson, of Wichita, Kans., July 25, 1931, at Wichita, Kans.

BIRTHS

To Dr. and Mrs. C. L. Davis, of Denver, Colo., a son, Richard Samuel, July 21, 1931.

To Dr. and Mrs. H. Glenn Ricker, of Waynesboro, Pa., a son, Carl Allison, July 30, 1931.

PERSONALS

Dr. R. O. Nye (Iowa '31) has located at Orion, Ill., for general practice.

Dr. Ed Everett (Ala. '23) reports a change of address from Auburn, Ala., to Leesburg, Fla.

Dr. R. C. Klussendorf (Corn. '31) has joined the firm of Downing and Lothe, at Waukesha, Wis.

Dr. J. A. Winkler (Cin. '18), of Fort Thomas, Ky., has been elected president of the Newport (Ky.) Rotary Club.

Dr. Thomas P. Nankervis (O. S. U. '31) has located at 423 Monroe St, Eveleth, Minn., where he will conduct a general practice.

Dr. Elvin R. Coon (Ind. '21), formerly of Logansport, Ind., has removed to Pittsfield, Mass. Address: 96 Howard St.

Dr. M. Jacob (U. P. '99), of Knoxville, Tenn., addressed the Knoxville Kiwanis Club, on August 6. His subject was "Rabies."

Dr. R. A. Miller (Ind. '17), of Indianapolis, Ind., has taken over the practice of Dr. H. J. Weaver (Ind. '17), at Hagerstown, Ind.

Dr. J. P. Torrey (Mich. '31) has accepted an appointment in the Laboratory of Animal Pathology and Hygiene, University of Illinois, Urbana.

Dr. S. R. Elkow (Ont. '23), of Highland Park, Mich., underwent an operation for appendicitis, in the Highland Park General Hospital, in August.

Dr. M. L. Boevers (Iowa '24) has left the Alabama Polytechnic Institute to accept a position in the Division of Veterinary Medicine, Iowa State College, Ames, Iowa.

Dr. W. A. James (O. S. U. '29) has resigned his position at the University of Illinois, to accept one with the Iodine Educational Bureau, with headquarters in New York City.

Dr. Cecil Elder (K. S. C. '16), who has been at the University of Wyoming for about twelve years, has left that institution to accept a position at the University of Missouri.

Dr. E. H. Barger (K. S. C. '21), formerly at the University of California, Berkeley, has accepted a position in the veterinary diagnostic laboratory at the University of Illinois.

Dr. Ernest F. Waller (Iowa '31), of Stewartsville, Mo., has accepted a position in the Veterinary Division, University of Minnesota, University Farm, Saint Paul, Minn.

Dr. Leon Abrevaya (Corn. '29) has accepted a position with the Minister of Agriculture of Venezuela. His headquarters are at Caracas. Dr. Abrevaya was formerly with the A. S. P. C. A., of New York City.

Dr. S. L. Stewart (K. C. V. C. '07), of Olathe, Kans., addressed the Kansas State Holstein Friesian Association, at Osawatomie, Kans., On July 22. Dr. Stewart's subject was milk conditions and milk production.

Dr. John S. Koen (K. C. V. C. 107), of Bloomington, Ill., has been appointed Chief Meat Inspector, City of Saint Louis, Missouri, and entered upon his new duties the latter part of August. (See JOURNAL, August, 1931, p. 216.)

Dr. William E. Jennings (Corn. '31), formerly at the New York State Agricultural Experiment Station, Geneva, N. Y., has joined the staff of the Division of Veterinary Medicine, Kansas State College, Manhattan, Kans.

Dr. O. T. Thomas (O. S. U. '28), of Pataskala, Ohio, has taken over the practice of the late Dr. W. E. Clemons, at Granville, Ohio. Dr. Thomas was formerly engaged in tuberculosis eradication with the Ohio State Department of Agriculture.

Dr. A. H. Dabbelt (Cin. '15), of Coldwater, Ohio, luckily escaped serious injuries, the evening of August 6, when his car collided with another, about two miles west of Coldwater. Dr. Dabbelt's car was badly damaged, but he escaped with minor bruises.

Dr. G. S. Muir (Ont. '28), of Wellesley, Ontario, will leave shortly for Camden Town, London, England, where he will spend a year at the Royal College of Veterinary Surgeons. Upon receiving his M.R.C.V.S., he will engage in general practice in England.